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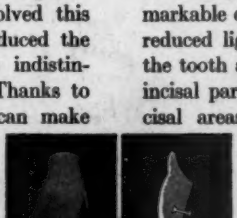

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THE REST POSITION OF THE MANDIBLE AND ITS SIGNIFICANCE TO DENTAL SCIENCE*

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It has been accepted as fact for many years that the relationship of the mandible to the maxilla is entirely dependent on the interlocking of the teeth in occlusion. This belief has led naturally to the conclusion that when the teeth are lost or severely abraded, the vertical dimension of the face is decreased. This apparently logical assumption has been founded on anatomic descriptions of the skull; for instance, that in Gray's Anatomy,[1] which states: "The most striking feature of the old skull is the diminution in size of the maxilla and mandible consequent to the loss of the teeth and the resorption of the alveolar processes. This is associated with a marked reduction in the vertical measurement of the face and with alteration in the angles of the mandible." Note that Gray refers to the "old skull" and not to the old living individual.

Another reason that the teeth have been given credit for establishing vertical dimension was the teaching that the gum pads were in approximation at birth (Fig. 1) and that, as the teeth erupted, the jaws were forced apart, with an increase in vertical dimension of the face. These misconceptions were not the result of incompetent or careless investigation, but rather of limitations imposed by the methods of study available at the time.

The effect of these teachings on dentistry has been the almost universal acceptance of the concept that the teeth contribute to the height of the face. This mistaken concept has led to numerous difficulties. For instance, dentistry has experienced an unscientific wave of "opening the bite" technics, which had for their object the restoration of supposedly lost or diminished vertical dimension. The warnings of Mershon,[2] Tench,[3] Schuyler[4] and others were ig-

*From the Department of Orthodontia, University of Illinois, College of Dentistry.

*First Prize Winning Essay, Fourth Annual Essay Contest of the Chicago Dental Society.

1. Gray, Henry: Anatomy of Human Body. Ed. 23, revised by Warren H. Lewis. Philadelphia: Lea & Febiger, 1936, p. 142.

2. Mershon, J. V.: Possibilities and Limitations in Treatment of Closed Bites. Internat. J. Orthodontia 23:581-599, June 1937.

3. Tench, R. W.: Dangers in Dental Reconstruction Involving Increase of Vertical Dimension of Lower Third of Human Face J.A.D.A. 25:566-570, April 1938.

4. Schuyler, C. A.: Problems Associated with Opening Bite Which Would Contraindicate It as Common Practice. J.A.D.A. 26:734-740, May 1939.

nored, and the enthusiasm for these procedures was dampened only by the large percentage of failures.

Some dental operations, based on sound biologic principles, have withstood the test of time with little or no variation from their original form. A case in point is G. V. Black's method of cavity preparation. This mechanical dental operation is based on the pathology of dental caries, the inclination of the enamel rods, the resiliency of dentin, the age of the patient and other anatomic and physiologic conditions.

Such soundness of principle is not evident in certain other fields of dentistry. In orthodontia, there is disagreement as to the nature of certain types of malocclusion, and consequently as to the method of treatment. For example, in Class II (Angle) malocclusion, one group advocates forward movement of the mandible, while another strives for distal movement of the maxillary teeth. In full denture prosthesis, we have a variety of methods for securing centric and vertical relations. Impression technics vary from the use of pressure to the advocacy of no pressure. Some believe

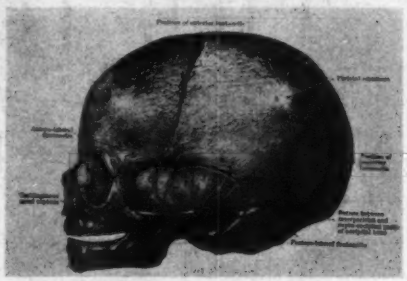

Fig. 1.—Generally accepted view of relationship of jaws at birth. (Gray's Anatomy, Fig. 156.)

that, in centric relation, the condyles are in the most retruded position in their fossae, while others maintain that they are not. Such diversity of opinion reflects a lack of knowledge upon which to base clinical procedures.

Until comparatively recently, no methods had been developed for obtaining scientific data on the positioning of the mandible in the living individual. Numerous clinical technics had been developed and advocated for establishing centric relation and paths of movement, but all of these were uncertain because

Fig. 2.—Left: Tracing of lateral head x-ray film of 3-day-old infant. The jaws are widely separated, the tongue filling the entire oral cavity. Right: Superposed tracings of head x-ray films at ages 3 months, 3 years and 8 years; showing orderly growth pattern. (Brodie.)

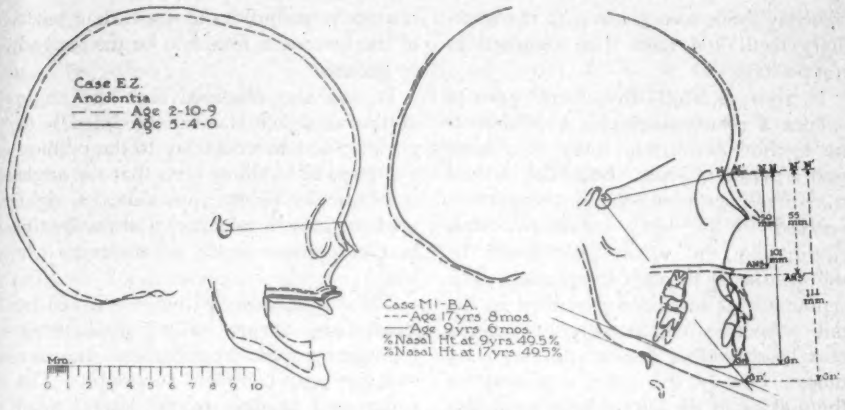

Fig. 3.—Left: Superposed x-ray tracings of child with complete anodontia. (Brodie and Sarnat.) Right: Superposed tracings made with mandible in rest position, on same child, at ages 9 years 6 months and 17 years 8 months. The facial proportions and relationship of mandible to maxilla are unchanged.

they required the placing of foreign bodies, such as biterims, in the mouth, and the employment of facebows. The presence of these encumbrances frequently gave rise to unnatural movements of the mandible, and hence results were uncertain.

In spite of the lack of an exact method, occasionally some one, through unusual gifts of perception, anticipated the scientific solution of problems, sometimes years in advance. This is true in the case under discussion.

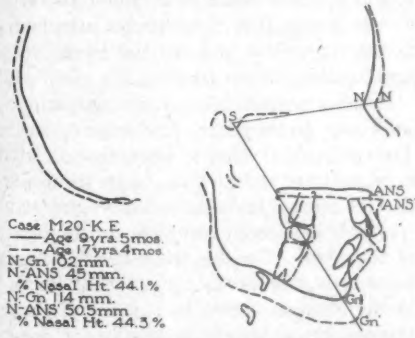

Fig. 4.—Superposed tracings of x-ray films made with mandible in rest position, on child referred to in Figure 3, at 9 years 5 months and 17 years 4 months. The facial proportions and relationship of mandible to maxilla are unchanged.

Niswonger[5] and Gillis[6] have long contended that the true vertical dimension of the face exists when the teeth are separated and the mandible is at rest. Niswonger defines it as "that position of the mandible in which it is involuntarily suspended by the reciprocal coordination of the muscles of mastication and the depressor muscles, with the upper and lower teeth separated, or the neutral position of the mandible." The simplest way to determine the rest position is to place the fingers over the temporal or masseter muscles and swallow. The muscles will be felt to contract and the teeth will come into occlusion. As the muscles relax, the mandible will move back to rest position. The mandibular teeth have traveled through the

5. Niswonger, M. E.: Rest Position of Mandible and Centric Relation. J.A.D.A. 21: 1572-1582, September 1934.

6. Gillis, R. R.: Establishing Vertical Dimension in Full Denture Construction. J.A.D.A. 28:430-435, March 1941.

free-way space to occlude with the maxillary teeth and have then returned to rest position.

In 1931, B. Holly Broadbent[7] gave to science a roentgenographic cephalometric method permitting study of a head over a period of years. Since the method is roentgenographic, the investigator is enabled to use bony landmarks, thus eliminating the errors introduced by the measuring through soft tissues. The cephalometer has been described in detail elsewhere. Essentially, it consists of a head-holding device and two x-ray tubes. One of the tubes is placed for the making of the lateral head x-ray film and the other for the frontal x-ray film. The successive films are strictly comparable, because the usual variables have been eliminated by standardizing the relationship of the tubes, films and subject.

Brodie,[8] who employed this method in his study of the growth of the head, observed that the growth pattern was established by at most 3 months of age. At birth, the jaws were found to be apart, with the tongue occupying the entire mouth cavity and extending over the ridges to support the lips. (Fig. 2, left.) With the eruption of the teeth and growth of the jaws, the tongue, growing at a slower rate, was gradually enclosed by the alveolar processes and the teeth, but at no time were the teeth in occlusion. This is contrary to the opinion that, as the teeth erupt, the jaws are forced apart. Figure 2, right, which represents tracings of headplates over a period of eight years, illustrates the orderly unfolding of an individual growth pattern. The floor of the nasal cavity is seen to descend on parallel planes, as does also the lower border of the mandible. In any given individual, the percentage contributions of the various parts of the face were found to be unchanged by growth.

It was also observed that the angle of the mandible was not modified with growth; which is contrary to the opinion expressed in anatomy texts that the angle is obtuse at birth, approaches a right angle as growth advances and eventually becomes obtuse again as the teeth are lost.

These observations were confirmed by Brodie and Sarnat[9] in a cephalometric roentgenographic appraisal of the case of a boy with complete anodontia. The superposed tracings of the lateral head x-ray films of this child taken at the age of 2 years 10 months and 5 years 4 months are shown in Figure 3, left. Despite the absence of all deciduous and permanent teeth, the mandible did not alter its form. Furthermore, it maintained a constant vertical and horizontal relationship with the maxilla and cranium. The facial proportions of this patient were well within the limits for the same values of the general population. Only the musculature with its growth pattern coordinated with the skeletal growth pattern could account for the maintenance of the mandibular position. It was assumed from these investigations on the living that the muscles attached to the mandible, and not the teeth, determine mandibular position.

Skeletal muscle has certain characteristics not possessed by any other tissue. The individual muscle fiber reacts "all or none" to stimuli; i.e., any stimulus that is strong enough to elicit any response brings about complete contraction of the fiber. On the other hand, when a study is made of a collection of fibers in the form of a muscle, it is found that the muscle is always in a state of partial contraction, even when resting. Thus,

7. Broadbent, B. H.: New X-Ray Technique and Its Application to Orthodontia. Angle Orthodontist 1:45-66, 1931.

8. Brodie, A. G.: Growth Pattern of Human Head from Third Month to Eighth Year of Life. Am. J. Anat. 68:209, March 1941.

9. Brodie, A. G., and Sarnat, B. G.: Ectodermal Dysplasia (Anhidrotic Type) with Complete Anodontia. Am. J. Dis. Child. 64: 1046-1054, December 1942.

we face the apparently contradictory conditions that, although the group of fibers may exhibit a state of partial contraction, the individual fibers of which it is composed can only be fully relaxed or fully contracted. The state of partial contraction exhibited by the resting muscle is known as tonus, and it is now known that the maintenance of this state is the responsibility of a few fibers, fully contracted, for short periods of time. As these fibers become fatigued, others take over the task, while the first group relaxes completely. The resting length of a muscle is its true length and the length to which it always returns after function. A muscle may be contracted, but it cannot be kept contracted. Similarly, as stretching forces are applied to it, resistance sets in, increasing as the stretching force is increased.

Since tonus implies tension within the muscle, it follows that if the muscle were not attached to anything, it would contract. That this is actually the case is shown by the shortening of the two ends of a cut muscle. Since the muscle is attached, the part to which it is attached tends to move in the direction of the muscle pull. That it does not move when at rest is due only to the fact that such movement is resisted by some other force which exactly balances it. This force is derived either from antagonistic muscles or from gravity, or from a combination of muscle force and gravity. Thus, the muscles around a joint have the dual function of (1) providing for movement and (2) maintaining stability in its resting position.

Movement of a joint in any direction involves the shortening of one set of muscles and elongation of the opposing or antagonistic set. Sherrington[10] explained how this interaction was possible by his demonstration of reciprocal innervation of muscles. The stimulation of one muscle group to contract causes the opposing muscles to relax proportionately, thus imparting smoothness to the movement. In a similar manner, it becomes apparent that, without the active and continuous contraction which is exhibited as muscle tonus, the posture and equilibrium of the body could not be maintained.

The application of these principles to the posture of the head reveals the reason for the stability of the mandible at rest. The vertebral column is essentially a rod composed of spools set one upon another. On top of this flexible column is the head. All sections are joined together by ligaments, but the erect position is maintained by the coordinated action of the muscles surrounding the column. Bilaterally, this balance is made possible by the equality of the muscles of the right and left sides in number, size and direction of action. The anteroposterior arrangement is more obscure. The head is positioned on the column in such manner that there is more weight anteriorly than posteriorly. This is evident in the forward nodding of the head when one falls asleep in an erect position.

The muscular antagonism to this forward tipping tendency of the head is derived from the powerful postcervical muscles, which run between the occipital bone and the column and thorax. Examination seems to indicate that these muscles are of a size out of proportion to a function of merely counterbalancing the pull of gravity and that, if this were their only source of antagonism, the head would be tipped back. Are there anterior muscular forces which, acting with gravity, balance the postcervical group?

A continuous anterior sheath of muscles from cranium to thorax would balance the posterior group, but would not permit mastication, deglutition or respiration. Each of these functions calls for

10. Denny-Brown, D.: Selected Writings of Sir Charles Sherrington. New York: Paul B. Hoeber, Inc., 1940.

the movement of different parts and, as a result, the anterior muscle arrangement is found to be in the form of a chain, of which the muscles of mastication, suprahyoid and infrahyoids, form the links. These links connect the mandible to the cranium, the hyoid bone to the mandible and the hyoid to the thorax, respectively, this making possible the independent, but coordinated movement of the parts. Thus, in deglutition, the masseter, internal pterygoid and temporal muscles contract to fix the mandible firmly against the maxilla. Thus, a stable base is afforded for the suprahyoid muscles to contract and elevate the hyoid bone. In order for the hyoid bone to move upward, the infrahyoid group must relax proportionately. At the same time, all three groups must be coordinated as a unit to maintain the posture of the head without nodding. The mandible and hyoid bone must be considered as connecting links in this muscular chain. A change in the position of either bone requires the shortening of some muscles and the lengthening of others.

Once the fact that the posture of the head is maintained by a series of antagonistic tensions and that these tensions cannot be altered except temporarily because of the physiologic properties of muscle tissue is recognized, the role of the mandible in the system becomes clear. Pulled upon from above by the tonus of the muscles of mastication and from below by that of the hyoid musculature and gravity, it can be at rest only when these tensions balance each other. Only when the mandible is in this rest position can we speak of the true vertical dimension of the face.

The vertical dimension of the face usually referred to in the literature is found only in the dried skull or when the muscles of mastication are contracted as in deglutition or in chewing. This vertical dimension will decrease with attrition if not compensated by continuous eruption, and of course it will decrease when teeth are lost; but neither of these events will affect the true rest position of the mandible: they will only affect the size of the free-way space between the jaws.

This essay presents the findings of investigations on the rest position of the mandible. These findings are offered as proof that the rest position is stable and unalterable and therefore affords the most accurate and reliable basis for analysis and treatment of a number of dental conditions. At the same time, it makes necessary acceptance of the fact that there are two positions of the mandible; i. e., the functional, in which the teeth are in occlusion, and the resting position, in which the teeth are not in occlusion. These two positions naturally give different vertical dimensions to the face, the occlusal vertical dimension and the rest vertical dimension.

Method

The method used in this study was roentgenographic, utilizing the Broadbent-Bolton cephalometer to position the head and standardize the x-ray procedure. The technic of obtaining a roentgenographic registration of mandibular rest position is as follows: The subject, seated in the chair with the head properly adjusted in the holder, is told to relax and be as comfortable as possible. He is engaged in conversation until tension is relieved, and is requested to swallow. A few seconds later, the exposure is made. No reference is made to the teeth or mandible, but rather the patient's thoughts are directed away from the procedure. Usually, the jaws will be in rest position unless the patient is instructed to bring the teeth together.

Findings

1. *Analysis of Rest Position in Growing Persons Over a Period of Years.*—In 1936, cephalometric roentgenograms

were made of a group of seventy-five children, in older age ranges than those studied by Brodie. At that time, the routine lateral exposure was taken with the teeth in occlusion. Some of the children were apparently not instructed to occlude the teeth, and the jaws were found to be separated when the films were processed. In 1944, additional records made on the same group included a lateral x-ray film with the mandible at rest.

The x-ray tracings of those children with teeth not in occlusion in the 1936 films were compared with the tracings of their recent rest position x-ray films. Figures 3, right, and 4 are superposed x-ray tracings of two of these cases. In Case M1-BA (Fig. 3, right), the angle formed by the projection of the lines N-S, representing the anterior cranial base, with the tangent to the lower border of the mandible at the age of 9 years 6 months, is 34 degrees. It was found to be unchanged at the age of 17 years 8 months. The proportions of the face remained constant, the nasal height (N-ANS) representing 49.5 per cent of the total face height (N-Gn) at both ages. In Case M20-KE (Fig. 4), the facial proportions were also unaltered. The linear dimensions naturally have increased with normal growth, but the facial proportions are the same. In not one of the seventeen children was there found a difference of as much as 1 per cent. Table 1 lists the percentages contributed by the nasal and dental areas to the total face height in the seventeen children who exhibited rest position at the time that the initial records were taken. The time interval between the first and second headplates, with the exception of Case 36, is approximately eight years.

The exact similarity of the facial proportions established with the mandible at rest indicates that the rest position has remained constant as growth progressed and that it is a part of the inherent growth pattern of the face.

2. *Analysis of the Rest Position in Children Undergoing Orthodontic Treatment.*[11]—A group of sixteen patients about to undergo orthodontic treatment were selected as subjects. In addition to the usual records, lateral head x-ray films were made with the mandible at rest. As the treatment progressed, additional rest position x-ray films were taken.

Since the subjects were growing children, the total face height with the mandible at rest must be expressed in facial proportions rather than in absolute dimensions. The percentage contributions of the nasal and the dental areas to the total face height were determined. A difference in the percentage in the later x-ray films was believed to indicate a change in the rest position of the mandible. The findings are shown in Figure 5. (See page 179.)

The column headed "Occlusion" lists the ratios of nasal height to total face height with the teeth in occlusion. The succeeding columns express the percentage of nasal height with the mandible at rest. The drop between occlusion and rest position, columns 1 and 2, is the amount of free-way space present. A straight line in columns 2, 3 and 4 indicates an identical position of the mandible at rest in successive x-ray films. In Cases NA, NC, NJ, NN, NO and NT, such a straight line was obtained. In Cases NG, NK, NL, NS, NU and T13, a slight variation was noted. Cases NB and NF exhibit a high degree of variation, explainable on the basis of use of intermaxillary elastics and headcaps in the orthodontic treatment. Both of these devices have long been known to tip the occlusal plane.[12]

11. Tockman, Irving; Asher, Carl, and Thompson, J. R.: Unpublished data, 1942.

12. Brodie, A. G., et al.: Cephalometric Appraisal of Orthodontic Results. Angle Orthodontist 8:261-351, 1938.

3. *Analysis of Rest Position of Edentulous Adults.*—Lateral cephalometric x-ray films were made of thirty edentulous adults with the mandible in rest position. Rest position was obtained in the same manner as that previously described. The measurements of the total face height, N to Gn (Fig. 6), are reported in Table 2. The number of x-ray films made for each patient is indicated by the number of measurements. The time interval between x-ray pictures varied from a few days to four years. Since growth was no longer active, linear measurements could be used.

In a few instances (second readings on 82, 93 and 94), there is a variation in measurements that would seem to contradict the contention that the mandible is stable in rest position. In 82 and 94, the first and third measurements are identical. Remarkable duplication is seen in all the other cases. The evidence of Cases 42, 67, 80, 85, 87, 134, 137 and 138 is particularly convincing since each series covered a number of years. The stability of the rest position is evident and is shown to be unrelated to the presence or absence of teeth.

4. *Cephalometric Analysis of Full and Partial Denture Cases.*—In 1939, a study was begun to observe the changes that occur after prosthetic restorations. Lateral x-ray films were taken before extraction, after extraction, before and after insertion of the dentures and at six-month intervals thereafter. The points and line of measurement of face height are shown in Figure 6. The program was initiated with ninety patients. Many of them did not respond to the recall letters; hence, adequate records are available for only one-third of the total. Thirty-five of these cases are reported in Table 4. To demonstrate the information compiled in this table, Case 65 is interpreted.

The first x-ray film showed the total face height with the mandible at rest to be 125 mm. With the dentures inserted and the teeth occluded, the occlusal vertical dimension was 122.5 mm. Twelve months later, this dimension was 122 mm., and it was found unchanged twenty-one months later. The rest vertical dimension of 125 mm. was confirmed by two additional x-ray films.

In those cases wherein the rest position was not exceeded and allowance was made for the free-way space, the occlusal vertical dimension did not decrease. Cases 31, 47, 65, 80, 88, 90 and 102 were constructed to the corrected occlusal height.

In a number of the cases examined, the occlusal vertical dimension has been unintentionally increased by the dentures to a point beyond rest position. This occurred in Cases 2, 30, 34, 39, 42, 67, 74, 83, 85, 87, 136 and 138. The clinical records revealed that these patients required more adjustment of their dentures than the previous groups did. In all but one of the cases (Case 67) in which opening is excessive, resorption of the supporting bone is resulting in a decrease. Two typical cases, 42 and 134, will be reported in detail. Neither of

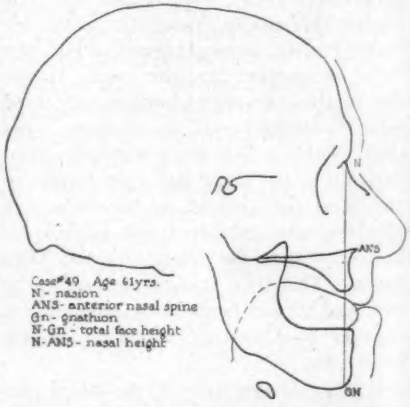

Fig. 6.—Tracing of edentulous case indicating points and line of measurement of total face height.

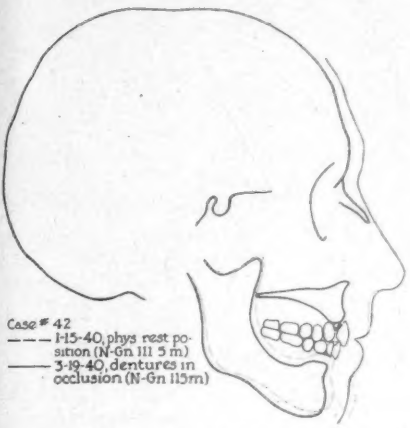

Fig. 7 (Case 42).—Superposed tracings of x-ray films before and after dentures were inserted. The bite has been opened excessively (6 mm.).

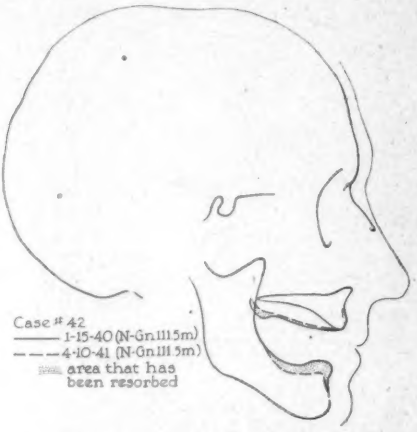

Fig. 8 (Case 42).—Superposed tracings of x-ray films illustrating resorption that occurred in thirteen months.

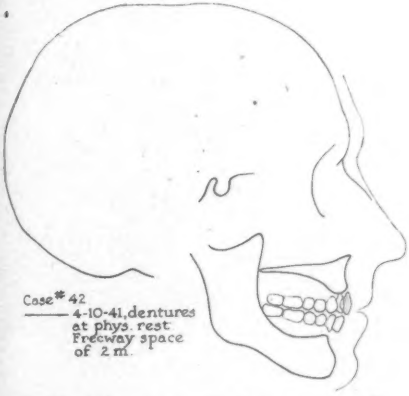

Fig. 9 (Case 42).—Tracing of x-ray films made thirteen months after dentures were inserted. The normal rest vertical dimension (111.5 mm.) has been reestablished and there is a free-way space of 2 mm.

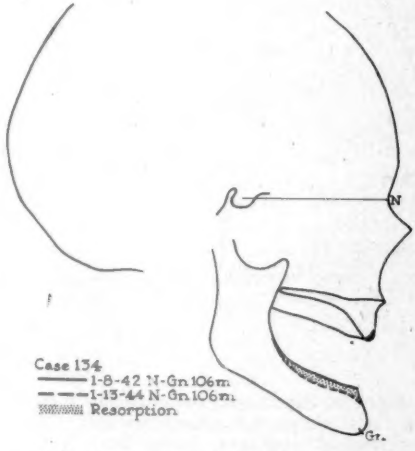

Fig. 10.—Tracing of edentulous x-ray films superposed on one made two years later showing bone resorption. The x-ray pictures are identical except for the area of resorption.

the patients had been subjected to recent extraction.

In Case 42, the rest position yielded a face height of 111.5 mm., a reading that was later confirmed by two other headplates. Figure 7 presents a tracing with the edentulous mandible at rest (N-Gn 111.5 mm.) superposed on that with the dentures in place (N-Gn 115 mm.). The bite had been opened about 6 mm. If we accept 111.5 mm. as the correct rest vertical dimension and allow 2.5 or

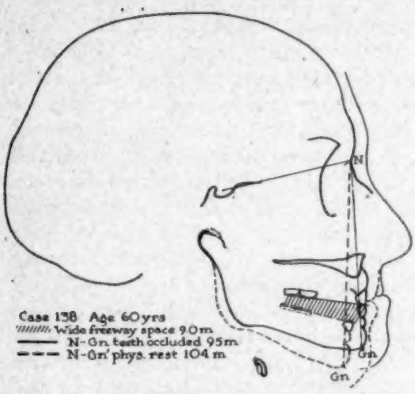

Fig. 11.—Wide free-way space, result of establishing deficient occlusal vertical dimension.

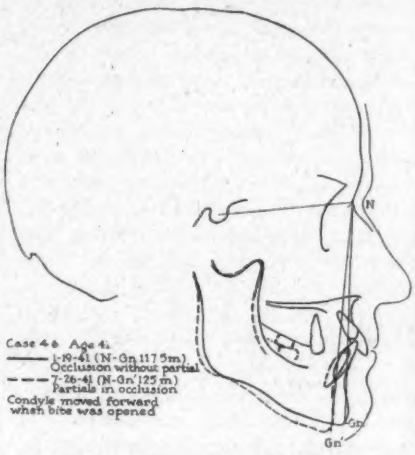

Fig. 12.—Occlusal vertical dimension increased from 117.5 mm., natural teeth in occlusion, to 125 mm., partial dentures in occlusion.

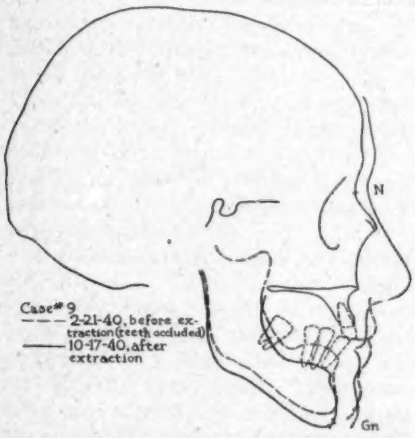

Fig. 13.—Superposed x-ray tracings showing facial change following extraction. The rest vertical dimension (solid line), eight months after the extraction, is greater than the occlusal vertical dimension (broken line). Facial change is not the result of decreased vertical dimension.

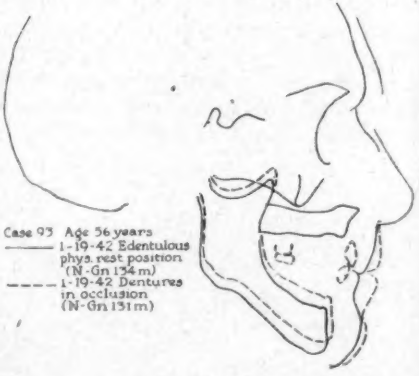

Fig. 14.—Changed facial contour following placement of full dentures. Occlusal vertical dimension with dentures in place is less than edentulous rest vertical dimension, but the lips are fuller.

3 mm. for the free-way space, the occlusal dimension should have been about 109 mm. In thirteen months, a considerable amount of resorption of the underlying bone occurred (Fig. 8). This closing of the bite not only permitted the mandible to assume its normal rest position, but also created a free-way space of 2 mm. (Fig. 9.)

In Case 134, the rest vertical dimension was found to be 106.5 mm. as determined by four x-ray examinations.

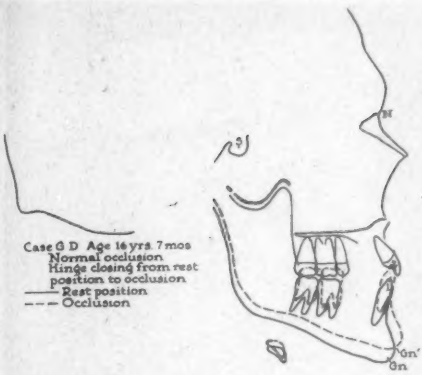

Fig. 15.—Normal path of closure of mandible from rest position to occlusion. Incisors and chin-point move upward and forward.

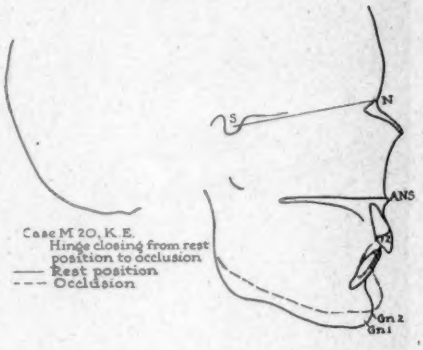

Fig. 16.—Normal path of closure of mandible from rest position to occlusion. Incisors and chin-point move upward and forward.

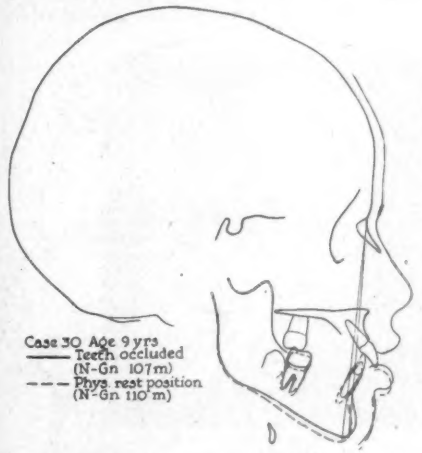

Fig. 17.—Normal path of closure in Class I malocclusion.

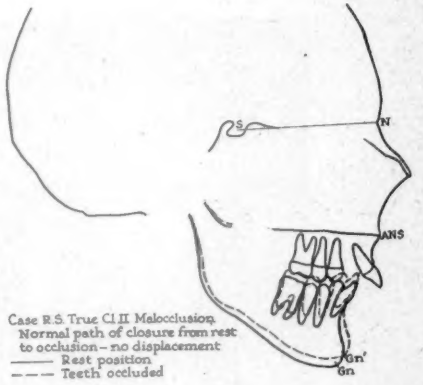

Fig. 18.—Normal path of closure in Class II, Division 1, malocclusion.

The dentures with the teeth occluded should have yielded a height of 103 or 104 mm. instead of the 111 mm. shown. Ten months later, this dimension had decreased to 105.5 mm., and in another year to 104.5 mm. The previous rest position was substantiated by additional rest x-ray films which measured 106, 106.5 and 107 mm. Figure 10 shows the resorption that occurred as the occlusal vertical dimension decreased to a level that was within the vertical dimension of rest.

Case 138 illustrates two important points concerning the free-way space. Should insufficient vertical dimension be established by the dentures, the free-way space will be excessive. (Fig. 11.)

Fig. 19.—Left: Models related in rest position. Right: Models in occlusion.

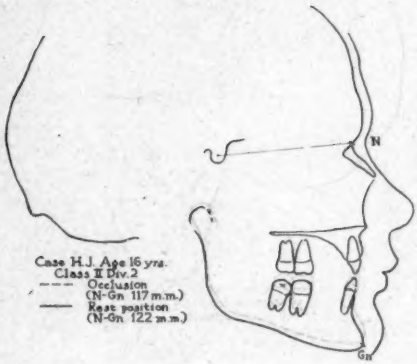

Fig. 20.—Superposed x-ray tracings of mandible at rest and with teeth in occlusion. The mandible is displaced upward and backward.

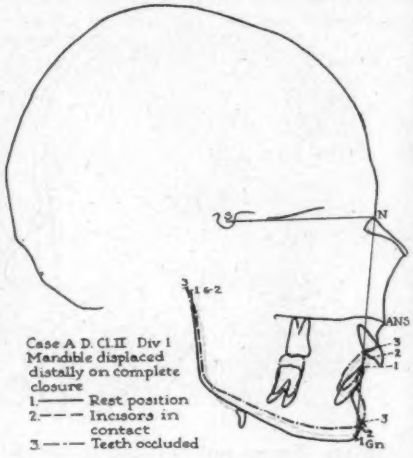

Fig. 21.—Superposed x-ray tracings of mandible at rest and in occlusion. The path of closure is normal from 1 to 2, but abnormal from 2 to 3.

This lessens the functional efficiency of the dentures because the closing muscles lose their power rapidly as they contract beyond the normal occlusal level. The second point to be noted is that demonstration of the usual amount of free-way space does not prove that the correct occlusal dimension has been secured. When the new denture was placed in this case, the bite was found to be opened to 102 mm. At the same time, a rest position of 105 mm. was recorded and a free-way space noted. This is an accommodation rest position, and

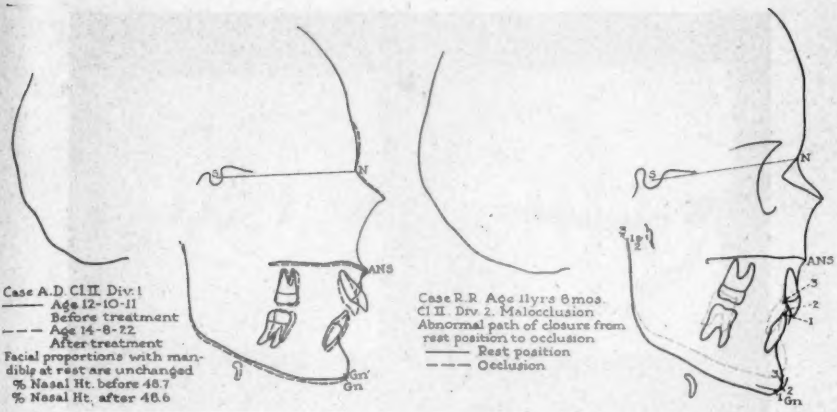

Fig. 22.—Left: Superposed x-ray tracings with mandible at rest before and after correction of malocclusion. The facial proportions are unchanged (difference of 0.1 per cent). Right: Superposed x-ray tracings of mandible at rest and teeth in occlusion. The mandible is displaced upward and backward.

Fig. 23.—Left: Models related in rest position. Right: Models in occlusion.

it is temporary. The occlusal dimension had closed to 99 mm. twenty-three months later, a level consistent with the normal rest dimension of 102 mm. Before leaving this case, the edentulous measurement of 100 mm. made the same day that the new dentures were placed requires clarification. This decrease in the rest measurement is the result of a temporary disturbance in muscle equilibrium, which can be reestablished only after the bite has closed to a level that is in harmony with the musculature. Case 136 also shows the temporary disturbance of rest position, but here again the occlusal dimension is decreasing by resorption of the bone supporting the dentures, so that muscle equilibrium can again be attained.

A full-mouth reconstruction in which all of the teeth have been built up with overlays has not been examined. From

Fig. 24.—Left: Models related in rest position. Right: Models in occlusion.

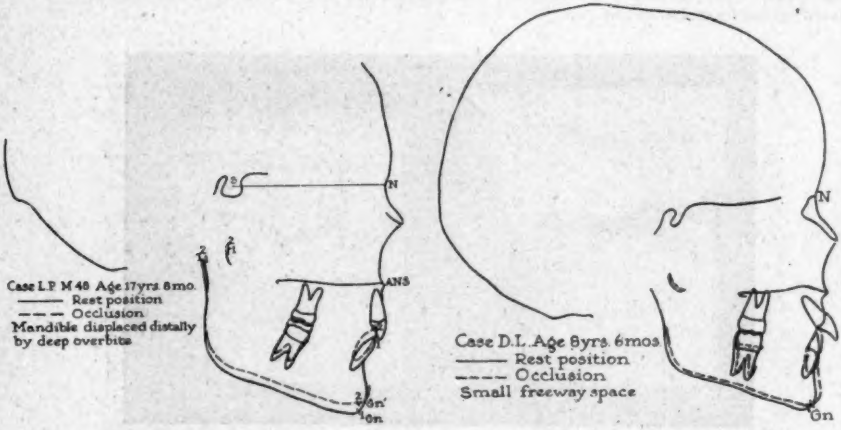

Fig. 25.—Left: Superposed x-ray tracings of mandible at rest and in occlusion. The mandible is displaced upward and backward. Right: Deep overbite associated with small free-way space.

the evidence presented, it would be expected that, if the rest position were exceeded by such a procedure, one or both of two conditions would arise. Either the teeth would be depressed or they would be loosened by the undue stress placed upon them by the increased tension of the closing muscles. (Breitner[13] increased the vertical dimension on a *Macacus mulatta* monkey with metal caps on the maxillary posterior teeth. The animal was killed after three weeks and the histologic examination revealed depression of the maxillary and mandibular molars with some traumatic changes.) That such risks are imminent

13. Breitner, C.: Further Investigations of Bone Changes Resulting from Experimental Orthodontic Treatment. Am. J. Orthodontics 27:11, January 1941.

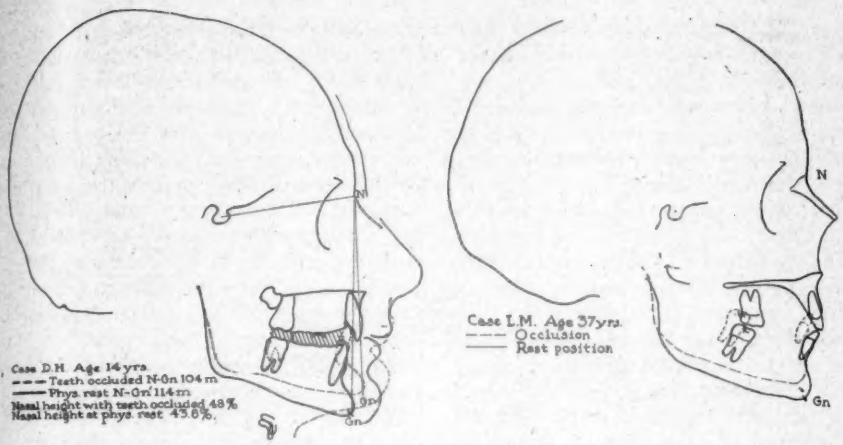

Fig. 26.—Left: Deep overbite associated with wide free-way space. Right: Superposed x-ray tracings with mandible at rest and in occlusion. The mandible is displaced upward and backward.

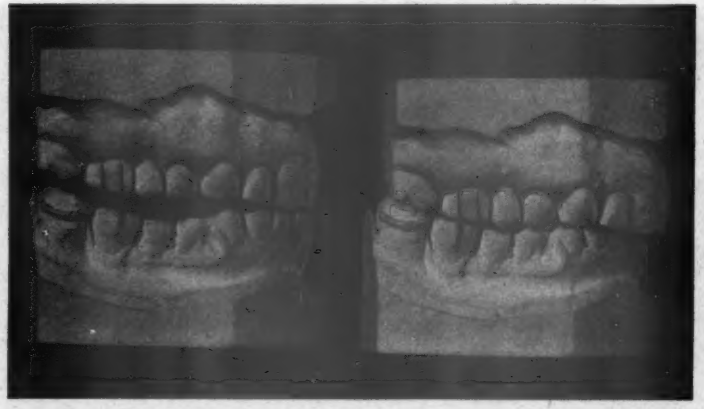

Fig. 27.—Left: Models related at rest position. Right: Models in occlusion.

under these conditions is clearly shown in the sequence of x-ray films for Case 46. The bite was opened 7.5 mm. by upper and lower partial dentures. The dates, measurements and status of the occlusion are listed in Table 3.

The occlusal vertical dimension established by the partial dentures was 125 mm., an increase of 7.5 mm. (Fig. 12.) Coincidentally with this increase, the patient began to complain of soreness of the muscles, clicking and subluxation of the left temporomandibular joint. These discomforts, often attributed to closed bite, continued during the time that the partial dentures were worn. In eight months, the occlusal vertical dimension decreased 2 mm. During this time, certain of the natural teeth elongated because they had been taken out of occlu-

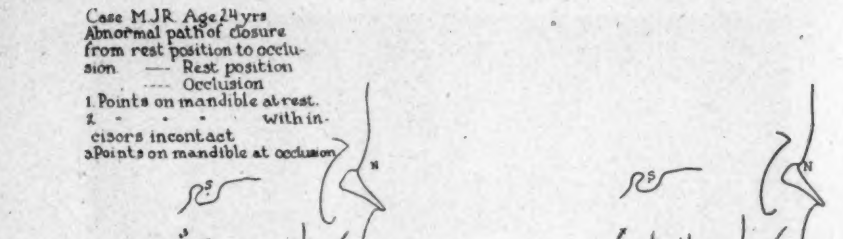

Fig. 28.—Left: Superposed x-ray tracings with mandible at rest and in occlusion. The path of closure is normal from 1 to 2. The mandible is displaced from 2 to 3. Right: Superposed x-ray tracings with mandible at rest, and splints and anterior teeth in occlusion.

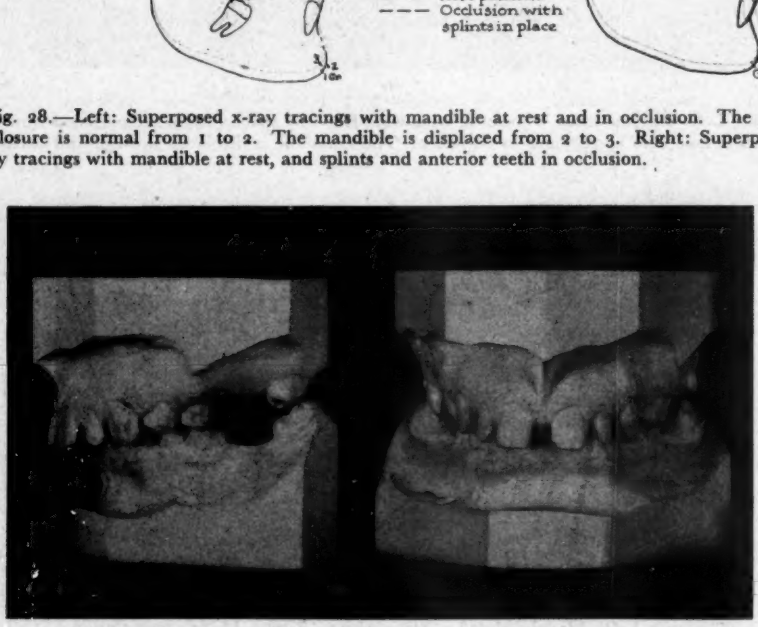

Fig. 29.—Left: Lateral view of teeth in occlusion. Right: Frontal view of teeth in occlusion.

sion by opening the bite. This increased the occlusal dimension of the natural teeth from 117.5 mm. to 121 mm. The case was reexamined twenty-one months later. The occlusal dimension with partial dentures in place was found to be 122 mm. and with only the natural teeth, 120. The teeth were extremely loose as they could not be depressed sufficiently to be relieved of the continual force of the extended musculature.

This serial study not only confirms the opinion that the rest position is stable, but also points out that it cannot be permanently altered by prosthetic restorations.

5. *Analysis of Facial Changes Following Loss of Teeth.*—Another mistaken

idea pertaining to vertical dimension concerns the facial changes following the loss of the teeth. It has been said that the "caved-in" appearance of the lips is the result of the chin approaching the nose, owing to a decrease in the vertical dimension. This clinical observation has been misinterpreted because of lack of an accurate method of appraising the condition. Figure 13 illustrates the typical facial change that occurs after the teeth are extracted. The vertical dimension associated with "sinking-in" of the lips (broken line) is greater than the occlusal vertical dimension (solid line) made before the teeth were extracted. The facial change is not the result of a decrease in vertical dimension, but rather of the loss of support given the lips by the teeth and alveolar processes.

That restoration of facial balance is not related to the vertical dimension is shown in Figure 14. Here again, the "caved-in" facial outline is associated with the edentulous or rest vertical dimension. This is greater than the occlusal vertical dimension established with the dentures in occlusion. The improved facial outline is the result of restoring lingual support to the lips with the artificial dentures.

The facial outline created by dentures is not a matter of restored vertical dimension, but one of labiolingual positioning of the teeth and of thickness of the labial plates of the artificial dentures.

The foregoing evidence has been presented to confirm the introductory statement that the rest position of the mandible is stable and unalterable. Since this was found to be true, rest position is the logical mandibular postion from which malrelation of the teeth and jaws can be analyzed. Such an analysis is based on two important observations: the path of closure from rest position to occlusion and the degree of closure or the size of the free-way space.

6. *Normal and Abnormal Path of Mandibular Closure.*—The path of closure has been examined in more than 100 patients of varying ages with normal or abnormal occlusion. The normal closing movement of the mandible from rest position to occlusion is a hinge movement with the fulcrum located in the lower half of the temporomandibular joint. The chin point and mandibular incisors move upward and forward. The mandibular molars move on a similar, but smaller arc since they are nearer the fulcrum. In most instances, there is no translatory movement of the superior surface of the condyle. Occasionally, slight movement is noted, and in these cases the closure is translatory, rather than hinge. If slight, this may still be considered normal.

The method of observing the path as normal or abnormal is as follows: Lateral head x-ray films are made with the mandible at rest and with the teeth in occlusion. Since the head is fixed in the head holder, the tracings of the cranium and upper face can be exactly superposed. By noting the course followed by the incisors, chin point and other mandibular points, and by manipulating the tracings to locate the fulcrum approximately, the normality of the path can be determined. Figures 15 and 16 show the normal path in two persons with normal occlusion.

The normal path of closure is not confined to normal occlusion and the determination of its normality is an important part of the analysis and classification of malocclusion. In Case 30 (Fig. 17), a Class I malocclusion, the normal path of closure is present. The teeth, although in positions of malocclusion, do not interfere with the normal closing movement of the mandible. The normal path is also observed in Case R. S. (Fig. 18), a Class II, Division 1, malocclusion. There is no displacement of the mandible, which indicates that this case is true

Class II malocclusion. The distal position of the mandible is a balanced muscular position both at rest and when the teeth are occluded.

The next few cases are characterized by an abnormal path of closure from rest position to occlusion. Because of the significance of the path of closure to the classification and analysis of malocclusion, reference must be made to the Angle classification, which has served as a guide to treatment as well as a means of classifying malocclusion since its introduction in 1899. It is based on the mesiodistal relations of the teeth, dental arches and jaws, as indicated by the mesiodistal relation assumed by the first permanent molars. It is important to remember that the classification requires that the teeth be in occlusion. A brief outline of the classification is given here for reference.

Class I. Arches in normal mesiodistal relations.

Class II. Lower arch distal to normal in its relation to upper arch.

Division 1. Bilaterally distal, protruding upper incisors.

Subdivision. Unilaterally distal, protruding upper incisors.

Division 2. Bilaterally distal, retruding upper incisors.

Subdivision. Unilaterally distal, retruding upper incisors.

Class III. Lower arch mesial to normal in its relation to upper arch.

Division 1. Bilaterally mesial.

Subdivision. Unilaterally mesial.

In Case H. J., the teeth occluded in relationships that would classify the malocclusion as Class II, Division 2 (Fig. 19, right). The headplates (Fig. 20) and models related at rest position (Fig. 19, left) show that this is actually a Class I malocclusion. At rest, the mandibular and maxillary incisors are almost end-to-end and a free-way space of 6 mm. exists between the posterior teeth. As the teeth are occluded, the mandible is displaced backward and upward. Another observation indicating displacement of the mandible is the abnormal pattern of the attrition. Considerable wear has occurred on the incisal edges of the mandibular incisors, but there is no accompanying attrition on the posterior teeth.

The analysis of the case from rest position suggested the following plan of treatment. The occlusion should be adjusted so that the normal path of closure could be effected. This requires correction of the deep overbite and an increase in the occlusal vertical dimension of the face by elongation of the posterior teeth. This plan was carried out successfully.

That a change in the rest position does not follow correct orthodontic treatment is shown in Case A. D. This case also exhibits a deep overbite and a wide free-way space and the mandible is displaced backward and upward (Fig. 21). In Figure 22, the tracings of the rest position x-ray film before treatment are superposed on the one made after treatment. The facial proportions vary only 0.1 per cent. If the treatment had increased the occlusal vertical dimension beyond the limits of rest position or had not made allowance for the normal free-way space, relapse would have been inevitable.

Cases R. R. (Fig. 23) and L. P. (Fig. 24) are both characterized by backward and upward displacement of the mandible, deep overbite and a wide free-way space. They require treatment that will increase the vertical dimension of the face and eliminate the deep overbite.

Recognition of the distal displacement of the mandible by occlusal interference explains a clinical observation frequently made by orthodontists. Occasionally, there will be an unusually prompt response to treatment, though the case appears to be as severe as others which respond in the usual manner. The cases

that respond promptly are those in which occlusal disharmony has forced the mandible into a distal-occlusal relationship. This condition denotes an unbalanced muscular position, and the mandible moves forward as occlusal interference is eliminated. Comparison of Cases R. S. (Fig. 18) and A. D. (Fig. 21) shows that two cases which appear to be similar when the teeth are occluded are quite different when analyzed from rest position. From these cases, the possibility of error in an analysis and classification of malocclusion based solely on occlusion of the teeth is evident. Analysis from both the rest position and occlusion reveals any displacement of the mandible. It has been shown that, in Class II malocclusion, the mandible may be distally from the normal at rest as well as when the teeth are occluded (Fig. 18). In this instance, the musculature is balanced and in harmony with the occlusion. There are cases that appear to be Class II when the teeth are occluded, but an analysis from rest position as well as occlusion reveals an abnormal path of closure. In these cases, the distal position is not a position in which there is muscle balance, but one dictated by the occlusal arrangement of the teeth. The analysis of malocclusion from rest position not only accurately appraises the anteroposterior and lateral position of the mandible, but the vertical relationship as well.

7. *Rest Position and Determination of Overbite.*—Clinical observation has led to the conclusion that excessive overbite may be due to supraclusion of the anterior teeth or infraclusion of the posterior teeth, or a combination of the two. No means of locating accurately the area at fault has been offered. Consideration of this problem from rest position shows clearly the nature of the malocclusion. The correlation of the amount of free-way space and the degree of overbite when the teeth are in occlusion was found to be significant in a study of twenty-eight children with malocclusion. The age, sex, occlusal and rest vertical dimensions and measurements, classification of malocclusion (Angle) and degree of overbite are given in Table 5. The overbite was classified as follows:

1. Slight.
2. Medium—mandibular incisors one-half covered by maxillary incisors.
3. Deep—mandibular incisors entirely covered by maxillary incisors.
4. Severe—mandibular incisors contacting or impinging on the soft tissues of the palate.

Table 6 shows the degree of overbite correlated with a small (0.5-2.4 mm.), medium (2.5-3.4 mm.) or large (3.5-7 mm.) free-way space. It is apparent, in most instances, that the free-way space increases with the degree of overbite. Among the deep overbite cases, there are four having a small or medium free-way space, and of the seven cases with severe overbite, one (D. L.) has a small free-way space. In all of these cases, the overbite is the result of overeruption of the anterior teeth (Fig. 25, right). Any orthodontic procedure that leads to an increase in the occlusal vertical dimension would result in relapse as the posterior teeth were depressed by the overextended musculature. In the eleven cases of deep and severe overbite with a large free-way space, the overbite is the result of insufficient eruption of the posterior teeth (Fig. 26, left). The occlusal vertical dimension of the face is deficient and requires elongation of the posterior teeth. This is not a "bite-opening" procedure, but rather a "filling-in" of the bite by reducing an excessive free-way space to a normal free-way space. If these patients were adults and orthodontic treatment were contraindicated, the amount of free-way space could be reduced by the use of occlusal splints

or gold onlays, thus increasing the occlusal vertical dimension.

Report of Cases

L. M., aged 37, had severe pains radiating from the temporomandibular articulation and maxillary sinus on both sides for a period of eight years. The jaw muscles were constantly fatigued and strained and the patient ground her teeth severely at night. Examination revealed that the lower right first and second bicuspids and first molar and left second bicuspid and first molar had been extracted. Attrition was more pronounced on the mandibular incisors and cuspids than on any other teeth. Lateral head x-ray films were made with the mandible at rest and with the teeth in occlusion. In rest position, the mandibular incisors were almost end-to-end with the maxillary incisors and a free-way space of 6 mm. existed between the posterior teeth (Fig. 27, left). The superposed tracings (Fig. 26, right) showed an abnormal path of closure, the mandible being displaced upward and backward, in eccentric occlusion.

The displacement of the mandible was the result of an incorrect occlusal relationship of the teeth. No restorations had been made after extraction of the lower first molars. The drifting of the second molars and the loss of three additional teeth altered the occlusion, forcing the mandible into an abnormal occlusal relationship. When a mandibular partial denture was constructed, the teeth were incorrectly arranged to occlude in an eccentric relationship. This was the result of analysis with the teeth in occlusion.

The rest position analysis showed the procedure necessary for restoration of the correct occlusal dimension. Part of the free-way space was filled in with acrylic splints on the posterior teeth. This prevented the displacement of the mandible. The pain, fatigue and tenseness of the musculature and the tooth grinding stopped within three days. There has been no recurrence in four years except a moderate degree of tooth grinding.

M. J. R., aged 24, complained of a tired and strained feeling of the jaw muscles and pain in the maxillary sinuses. She had frequent dizzy spells and complained of a "stuffiness" in the ears. Examination by a physician revealed that the ears and sinuses were normal.

With the teeth in occlusion, this case would have been classified as a Class II, Division 1, malocclusion (Fig. 29, left). The overbite was severe, the mandibular incisors impinging on the soft tissues of the palate. A wide space between the maxillary incisors had become progressively larger. The occlusion had been altered by the loss of all four first molars, three second molars and one bicuspid.

Lateral head x-ray films were made with the mandible at rest, with the incisors in contact, and with the molars in occlusion. Superposition of the tracings (Fig. 28, left) showed the path of closure to be normal until the incisors occluded. The mandible was then displaced upward and backward into an incorrect occlusal position. The loss of the molar teeth had permitted the remaining teeth to drift and the lower anteriors to overerupt. As the decrease in the occlusal vertical dimension and displacement of the mandible became more severe, the subjective symptoms developed. (See Fig. 32.)

Occlusal acrylic splints were constructed to supply posterior occlusion at the same level of contact as that of the incisors—the termination of the normal path. Figure 28, right, shows the superposed tracings of rest position and the correct occlusal level, established by use of the splints. The path of closure was normal. The abnormal muscle tension was immediately relieved upon placing the splints. For three months, there has been no recurrence of pain, dizziness, muscle fatigue or discomfort in the ears.

The history and method of treatment of these two cases are not unusual and many similar reports are found in the dental literature. They demonstrate a method of analysis which accurately determines the degree of overclosure and the type of mandibular displacement.

Clinical Technics for Determination of Mandibular Rest Position

1. *Roentgenographic Method.*—For a roentgenographic registration of rest position, it is unnecessary to employ the highly accurate device with which these

Fig. 30.—Models with splints in occlusion.

studies were made. The dental x-ray machine will answer provided certain conditions are met. A target distance of least 3 feet must be employed to minimize distorted enlargement. Cassettes, 8 by 10 inches, equipped with fast, high definition screens and a three-second exposure (110 volts, 10 milliamperes) will yield satisfactory detail. The midsagittal plane of the head and the cassette must be parallel, and at right angles to the central ray axis. The patient should be seated in an erect, but comfortable position and should be cautioned not to move while the exposure is being made.

A series of exposures taken according to the foregoing directions will probably vary in size because the target distances will vary. This need cause no concern as absolute measurements are not necessary. The images will be proportional to the distances at which they were made, and therefore it is only necessary to reduce the measurements in proportion. The method is as follows: After tracing the headplate on a good grade of tracing paper, a line is drawn from the frontonasal junction (N) to the chin-point (Gn). The latter is determined by taking a point midway between the most anterior and inferior points on the bony chin. A perpendicular is then erected from the line just drawn to the top of the anterior nasal spine. This perpendicular divides the line representing face height into two parts, N-ANS, which represents nasal height, and ANS-Gn, which represents lower face height. The percentage contribution of nasal height is then computed by dividing the nasal height by the total face height.

Figure 31 shows the tracings of lateral x-ray films in one case, without the head holder, at target distances of 3, 5 and 7 feet. The absolute total face height ranges from 127 mm. at 7 feet to 132.5 mm. at 3 feet. The percentage contributions of the nasal height (N-ANS) to total face height (N-Gn) for the three distances are, respectively, 42.5, 42.7 and 42.6. The differential in values represents a discrepancy of less than 1 mm. The variation between the lines representing the right and left sides of the mandible shows that the head and film were not positioned identically for the three exposures. The similarity in facial proportions indicates that some error in the positioning of the head and cassette are permissible.

The technic just described is that recommended for the determination of mandibular rest position. Obviously, the patient must be told to relax and shown how he can do so by swallowing several times and noting how the mandible tends to return to a resting position. An excellent check is provided by repeating the exposure with a time interval of several days between sittings.

In order to study the path of closure of the mandible, it is necessary to make two exposures at the same sitting and with a minimum of movement between the two. This can be done by double-exposing the same film, exposing for the rest position first, and then asking the patient to slowly bring the teeth into occlusion without moving the head and making the second exposure. Exposures for the two positions may be made on

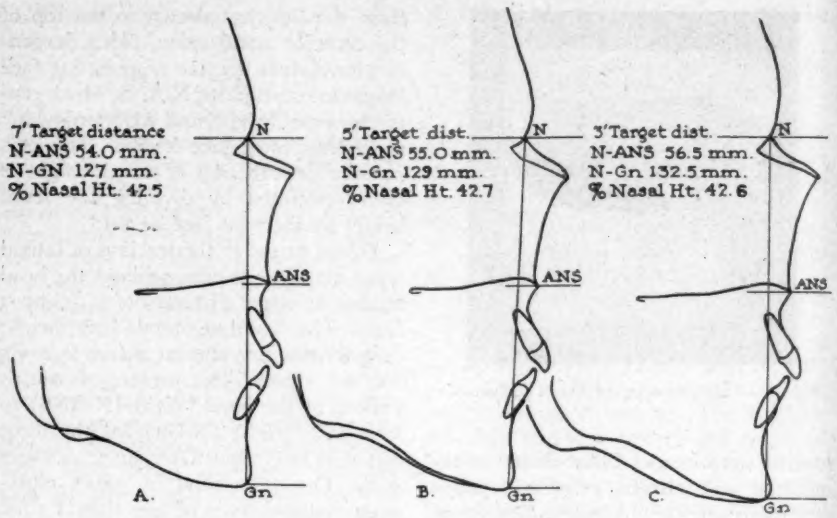

Fig. 31.—Tracings of three lateral x-ray films made without headholder at target distances of 3, 5 and 7 feet. The absolute size varies, but the proportions of the face are relatively the same.

separate films, of course, but will involve the use of two cassettes and a greater danger of movement and incorrect placement. With either method, each position should be traced on a separate piece of paper and the tracings subsequently superposed.

2. *Mechanical Method.*—The value of rest position in the analysis of malocclusion and abnormal positions of the mandible led to an attempt to develop a method by which plaster casts could be related in rest position and mounted on an articulator. It was found possible to do this by means of a plaster core.

A. Obtaining Rest Position: The patient is seated in a comfortable and relaxed position. Rest position is secured after speech and checked a number of times until the operator has a mental picture of the relationship of the incisors at rest. The patient is then requested not to move the jaws.

B. Insertion of Plaster: The lips are slowly parted and rest position is checked. A rapid setting mix of impression plaster is inserted between the lips and gently pressed over and between the anterior teeth. After this mix has set, additional plaster is placed in the buccal vestibules and forced into the free-way space by compressing the cheeks. When the plaster has set, the core is removed.

C. Trimming of Plaster Core: The roughened core is trimmed so that the cervical one-third of the teeth will be uncovered when the casts are mounted in the cores. (Figure 34, left, shows the unassembled cores after trimming.) Trimming insures accurate seating of the casts in the core.

D. Assembling Plaster Core and Casts: The core and casts are assembled and sealed with sticky wax. (Fig. 34, right.)

E. Mounting Casts on Articulator (Fig. 35): The type of articulator used is a matter of personal preference. The larger and more complex articulators will

Fig. 32.—Left: Profile with teeth in occlusion. Right: Profile with mandible at rest.

Fig. 33.—Left: Relationship of teeth at rest position. Right: Plaster core.

permit approximate demonstration of mandibular displacement. Regardless of the type used, exact duplication is not possible, nor is it necessary. It is sufficient to know that displacement of the mandible occurs in a given case. Face-bow registrations, while not used here, are desirable in order to relate the casts to the temporomandibular joints. The movement of the mandible from rest position to occlusion, essentially a hinge movement, can be reproduced on the articulator by releasing the lock or pin to permit the casts to move together a distance of 3 to 3.5 mm. (the average free-way space at the incisors). The in-

Fig. 34.—Left: Unassembled core after trimming. Right: Upper and lower casts mounted in core.

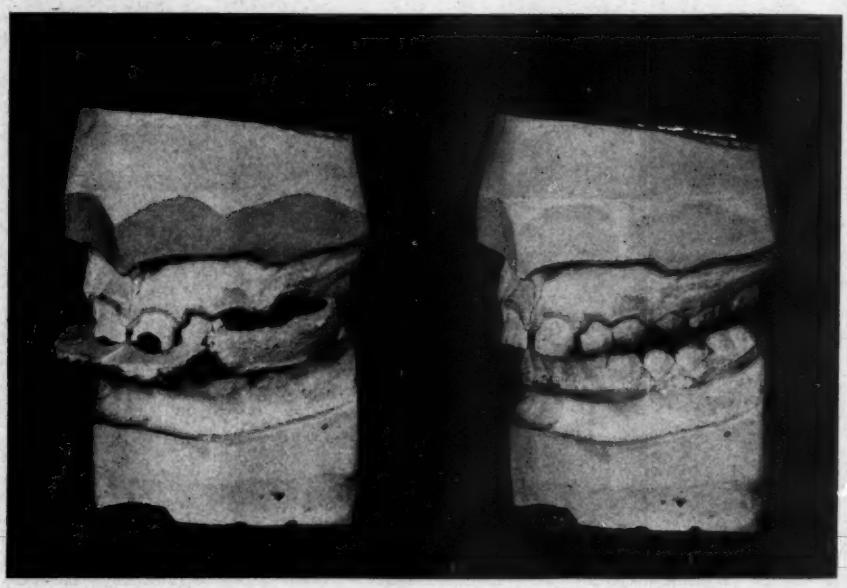

Fig. 35.—Left: Plaster core and casts mounted on articulator. Right: Rest position relationship of casts on articulator.

cisors may be in contact while a free-way space still exists between the posterior teeth as in Case M. J. R. (Fig. 28, left), or none of the teeth may occlude until overclosure occurs. These cases will permit occlusal reconstruction. On the other hand, certain teeth may encroach on the normal free-way space and offer points of interference. These may be removed by occlusal adjustment.

Observation of the casts mounted in rest position (Fig. 35, right) readily indicates the orthodontic or other treatment necessary to eliminate occlusal interference and to restore the occlusal vertical dimension.

Table 1.—Constancy of Facial Proportions with Mandible at Rest

Case	First Head Plate		Second Head Plate	
	% Nasal Ht. and Age	% Dental Ht.	% Nasal Ht. and Age	% Dental Ht
1	49.5 9 yr. 6 mo.	50.5	49.5 17 yr. 8 mo.	50.5
4	46.2 7 yr. 7 mo.	53.8	46.2 14 yr. 7 mo.	53.8
6	42.7 5 yr. 10 mo.	57.3	42.8 14 yr. 9 mo.	57.2
7	40.7 6 yr. 4 mo.	59.3	40.0 14 yr. 1 mo.	60.0
10	45.6 7 yr.	54.4	46.2 15 yr. 5 mo.	53.8
18	42.6 5 yr. 6 mo.	57.4	42.4 13 yr. 8 mo.	57.6
20	44.1 9 yr. 5 mo.	55.9	44.2 17 yr. 4 mo.	55.8
21	43.0 9 yr. 6 mo.	57.0	42.9 17 yr. 3 mo.	57.1
29	42.0 6 yr.	58.0	42.1 14 yr. 5 mo.	57.9
36	40.6 3 yr. 6 mo.	59.4	40.5 4 yr.	59.5
43	42.6 6 yr.	57.4	42.8 13 yr. 7 mo.	57.2
48	43.4 9 yr. 6 mo.	56.6	43.7 17 yr. 8 mo.	56.3
51	39.0 3 yr. 6 mo.	61.0	39.2 10 yr. 5 mo.	60.8
52	46.6 9 yr.	53.4	47.1 16 yr. 6 mo.	52.9
59	43.5 8 yr.	56.5	43.3 15 yr. 5 mo.	56.7
62	42.8 8 yr.	57.2	43.1 15 yr. 10 mo.	56.9
69	43.8 8 yr.	56.2	44.3 15 yr. 7 mo.	55.7

Summary and Conclusions

1. Dental science has been hampered by the teaching that the teeth contribute to the height of the face. This concept was based on examination of skulls and plaster models with the teeth in occlusion.

2. This concept has led to the assumption that as dental height diminishes through attrition or loss of teeth, there is a corresponding loss in height of the face. This loss has been held responsible for sinking in of the lips in the edentulous.

3. The application of the foregoing concept and its corollary to clinical practice takes the form of bite-raising technics which have for their object the restoration of supposedly diminished vertical height.

4. A long series of scientific investigations on the living have shown that the prevailing concept is incorrect. These studies have been conducted on large series of children from birth to 8 years, on children from 8 years through adolescence, on children with complete anodontia and on edentulous adults. Without exception, it has been shown that the mandible assumes its positional

Table 2.—Total Face Height (N-Gn) in Edentulous Persons

Case	1	2	3	4	5
30	107*	106.5	107	106.5	
31	118	117.5			
33	133	132.5			
34	119	120	118	120	119
39	126	128	128		
42	111.5	111.5	111.5	111.5	
47	122	122.5	122.5		
48	141	141			
52	131	131.5			
65	125	125			
66	120	118.5	118.5		
67	112	112	113	112	
70	127	128			
71	109	109			
74	120.5	120.5	121		
76	123	123			
78	101	108			
80	116	118	118	118	118
82	136	132	136		
83	128	127.5			

Case	1	2	3	4	5	6	7	8
85	115	115	115	115				
86	127	127						
87	127	126	126	126	126	126	126	
90	125	125						
93	134	131	135					
94	129	124	129	127				
102	124	124.5	124					
103	135	136						
109	110	110						
134	106.5	106.5	106.5	106	106	106.5	106.5	107
137	106.5	106	106.5					
138	102	102	102	102	102	102	102	

*Measurements of total face height (N-Gn) in millimeters.

Table 3.—Data from Case 46: Upper and Lower Partial Dentures

Date	N-Gn Mm.	Occlusion
1/19/41	117.5	Natural teeth occluded
7/28/41	125	Partial dentures occluded
3/13/42	123	Partial dentures occluded
3/13/42	121	Natural teeth occluded
1/21/44	120	Natural teeth occluded
1/21/44	122	Partial dentures occluded

*Table 4.—Full Denture Restorations**

Case	1	2	3	4	5	6	7	8	9	10	11	12
		2		2	14							
30	107	106.5	107	108.5†	104†	106.5						
		3	1	1	11							
34	119	121	118	129†	125†	122	119					
		2			21							
39	126	128	131	126†	124.5†	128						
		3	4	9								
40	116	118	116†	115†	120							
		2	13									
42	111.5	115†	109†	111.5	112	111.5						
		3	3									
48	141	141	146†									
			12	21								
65	125	122.5†	122†	122†	125	125						
				11	8			8				
67	112	112	117.5†	117†	117†	117†	113	111.5				
				9	8							
70	129	127	129†	129†	129†	129†	129					
		7	4				23					
74	120.5	122†	119†	120.5	119	120.5	119†	120.5				
		22	4			33						
76	127‡	123	124†	129	129	116†	123					
		3	11		8							
80	116	118	116†	118	116†	118	118					
		2	2									
82	136	132	136	135†								
			24									
83	127†	128	126.8†	127.5								
		2		21		33						
85	115	115†	111	114†	115	112†	115	115				
		4	10			22				12		
87	127	126†	124†	126	126	124†	126	126	126	124†	126	
		8	1									
90	122.5‡	125	125	122.5†								
		5	17									
93	134	133†	125†	131	134.5							
		8		8								
94	129	124	129	122†	128							
			¼		1	1		10				12
134	106.5	106.5	106.5†	106	111 †	111†	106	105.5†	106.5†		107	104.5†
			3			6						
136	111.5#	114.5	114.5	119†	119	115†	118					
			1			1			23			
138	95#	104	102	102	102	102†	105	100	99†	102	102	102
Case	1		2	3	4		5	6		7		8
		4		9								
2	135	136†		134.5†	136							
		2		15			31					
31	116‡	116†		116†	118		116†	117.5				
		6		8			22					
46	117.5‡	125†		123†	121‡		122†	120‡				
		2		8	5					8		
47	120.5	122		120.5†	120†		122.5	122.5		120†	122	
		4		10								
86	125‡	127†		125†	127							
		7										
88	111.5‡	113.5		111.5†								
		1		2			22					

102	124*	124.5	121.5 † (34)	121.5†	121.5†	124
110	124‡	128† (2)	124†	125		
113	117‡	117†	117†			

*Measurements of total face height (N-Gn) in millimeters. The numeral in upper left hand corner of box indicates the number of months that have elapsed since the preceding x-ray film was made.

†Full or partial dentures in occlusion.

‡Natural teeth in occlusion.

#Old dentures in occlusion.

Table 5.—Degree of Free-Way Space

	Age	Sex	Occlusal Vertical Dimension Mm.	Rest Vertical Dimension Mm.	Classification (Angle)	Degree of Overbite
TC	8-1-12	♂	108	114	I	Slight
TA	14-7-12	♂	116.5	120.5	I	Slight
TJ	30	♀	117	119	II, Div. 1	Slight
NG	14-6-10	♀	129.5	131.5	I	Slight
NH	12-7-1	♀	118.5	120	I	Slight
TG	10-3-5	♀	105	106	II, Div. 1	Medium
TF	17-8-4	♂	125.5	128.5	II, Div. 2	Medium
TD	11-7-21	♂	114.5	118	I	Medium
NI	12-6-25	♂	118.5	121.5	II, Div. 1	Medium
NF	11-7-13	♀	113	120.5	I	Medium
NO	12-11-27	♂	120	125	I	Medium
NR	15-1-3	♀	109.5	113	I	Medium
NP	11-3-18	♂	117.5	123.5	I	Deep
TH	13-11-17	♀	114.5	120.5	II, Div. 1	Deep
TB	12-2-28	♀	105	110.5	I	Deep
NX	17-9-13	♂	111.5	115.5	I	Deep
NL	14-6-29	♀	110.5	114	I	Deep
TK	11-5-7	♀	118	121	I	Deep
NT	11-8-9	♂	120	123	I	Deep
NA	11-7-3	♀	105.5	108.5	II, Div. 1	Deep
NJ	13-2-28	♀	122	122.5	II, Div. 1	Deep
TH	11-11-19	♀	106.5	112.5	II, Div. 1	Severe
NS	11-2-1	♂	113	118	II, Div. 1	Severe
NN	13-2-29	♀	111	116	II, Div. 1	Severe
NM	16-9-2	♂	120	125	II, Div. 1	Severe
NC	10-6-24	♂	106.5	110.5	I	Severe
NB	12-11-6	♂	110	115.5	II, Div. 1	Severe
DL	8-6-3	♂	97.5	100	II, Div. 1	Severe

Table 6.—Degree of Overbite Correlated with Free-Way Space

Slight Overbite

Small Free-Way Space
(1) NH (1.5 mm.)
(2) NG (2 mm.)
(3) TJ (2 mm.)

Large Free-Way Space
(1) TA (4 mm.)
(2) TC (6 mm.)

Medium Overbite

Small Free-Way Space
(1) TG (1 mm.)
(2) NV (2 mm.)

Medium Free-Way Space
(1) TF (3 mm.)
(2) NT (3 mm.)

Large Free-Way Space
(1) TD (3.5 mm.)
(2) NR (3.5 mm.)
(3) NO (5 mm.)
(4) NF (7.5 mm.)

Deep Overbite

Small Free-Way Space
(1) NJ (0.5 mm.)

Medium Free-Way Space
(1) NA (3 mm.)
(2) NT (3 mm.)
(3) TK (3 mm.)

Large Free-Way Space
(1) NL (3.5 mm.)
(2) NX (4 mm.)
(3) TB (5.5 mm.)
(4) TH (6 mm.)
(5) NP (6 mm.)

Severe Overbite

Small Free-Way Space
(1) DL (2.5 mm.)

Large Free-Way Space
(1) NC (4 mm.)
(2) NB (5.5 mm.)
(3) NN (5 mm.)
(4) NM (5 mm.)
(5) NS (5 mm.)
(6) TN (6 mm.)

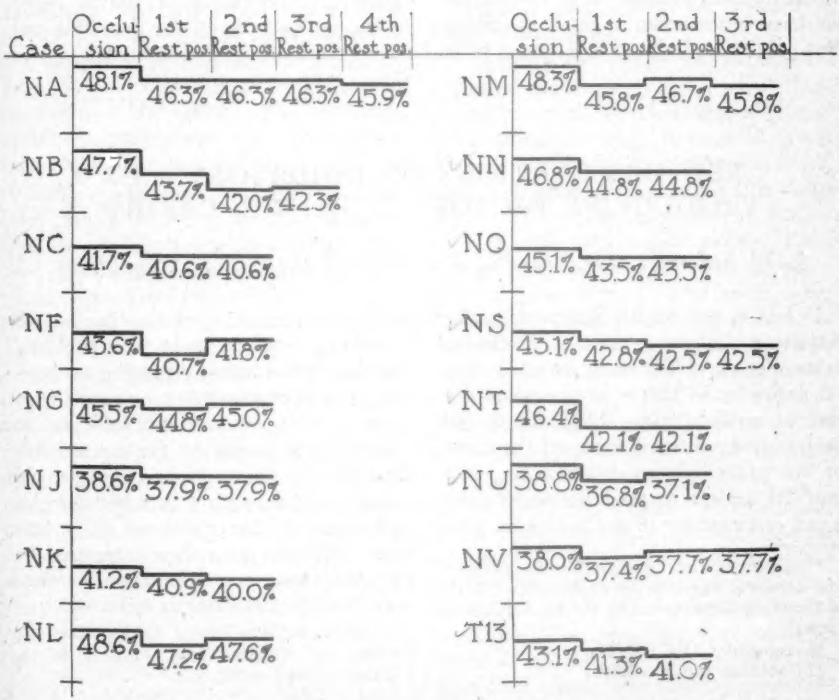

Fig. 5.—Analysis of rest position in patients undergoing orthodontic treatment.

relationship to the head by the third month of life and thereafter does not change.

5. The position here referred to is a resting position determined by a balance of the musculature which suspends the mandible. It is not affected by either the presence or the absence of teeth. This position alone determines the height of the face.

6. The difference between the height of the face when the mandible is at rest and when the teeth are in occlusion is determined by the amount of space between the upper and lower teeth when the mandible is at rest. This, which is called the free-way space, averages 2-3 mm. in normal dentition. In abnormal, it may be 10 mm. or more.

7. All attempts to increase the vertical dimension of the face beyond that established by the rest position of the mandible have been shown to result in failure. Not only does the mandible return to its resting position, but it invariably establishes a normal free-way space as well.

8. The normal closing path of the mandible from resting to occlusal position is of a simple rotary type, the condyle acting as the center.

9. In some persons, the path of closure is found to be abnormal because of occlusal interference. In these cases, the mandible may be displaced during deglutition and mastication. If the free-way space is larger in such a case, the mandible may overclose to a considerable extent, with resultant severe discomfort. Such cases may be relieved by filling in the free-way space to within the normal 2-3 mm. and by eliminating occlusal interference.

10. In accordance with these findings, the analysis of cases involving extensive restorative procedures and the classification of malocclusion should be based on the rest position of the mandible and not on the occlusion of the teeth.

NEW OBSERVATIONS ON INHIBITORY AND PRODUCTIVE FACTORS IN DENTAL CARIES*

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Defeat is not readily accepted by the American people; yet our profession acknowledges defeat when we allow dental caries to be the most prevalent disease of civilized man. Shall the dental profession continue to write off the cause of this pathologic condition as an enigma? In answer to this challenge, there is not one member of the profession who has not at some time or another paused to reflect on this, our own great problem. Current dental literature indicates, however, that strangely few investigators were inspired with a conviction sufficient to warrant new studies on the matter. Additionally, in the published reports, not a single author, with a firm belief in his investigations, has produced truly new ideas, different from those expressed by an anonymous communication in German literature as early as 1530 A.D.[1]:

*"Assertions and opinions are the authors' and do not in any way reflect the official views of the Navy Department or the U. S. Navy at large."

†Commander (DC) USN.

‡Lieutenant (DC) USNR.

1. Kesel, R. A.: Dental Caries: Etiology, Control and Acidity Tests. J.A.D.A. 30:25, January 1, 1943.

Caries is a disease and evil of the teeth in which they become full of holes and hollow, which most often affects the back teeth, especially so when they are not cleaned of clinging particles of food which decompose, producing an acid moisture (literally, a sharp moisture) which eats them away and destroys them so that finally with much pain they rot away little by little.[2]

This thesis, then, will be devoted to a new approach, with a postulate, based on our experiments, that the action of certain bacteria and fungi in dental caries is inhibitory or stimulative, depending on which of the organisms is predominant in the saliva.

Evidence has been presented in current literature that salivary factors do influence dental decay. Because dental decay is a prevalent disease peculiar to man and not found generally in lower animals, it is presumed that the saliva of the caries-free does contain some buffering or retarding agent which prevents dental caries. Inversely, it may be possible that the saliva of the caries-susceptible contains some agent which is a factor in acute caries.

Since research on dental decay has been conducted for many years and no new theory regarding its etiology has been presented, it was decided that the search for causes should be waived for the present, and an approach be made from observations of the processes accompanying formation of dental cavities, in an attempt to devise effective countermeasures. The employment of organic products, such as endocrine substances obtained from nonsusceptible persons and other biologic and therapeutic agents used in this form of research, opens an avenue for experimentation. That this method is acceptable is evidenced by examples from other fields. The cause for inactivity of the islands of Langerhans is not known, but many persons suffering from diabetes are able to live normal lives by daily injection of prepared insulin. Likewise, the therapeutic value of cod liver oil was recognized long before vitamins were discovered.

It was decided that, to obtain preliminary information, the action of various organic and therapeutic substances on tooth structure *in vitro* should be observed. By this method, we hoped to demonstrate that tooth structure submitted to the action of saliva obtained from subjects in whom caries was rampant would lose more weight than tooth structure submitted to the action of saliva from the caries-free. It was also hoped that, in this approach, it would be possible to detect the presence of inhibitory and stimulative agents in the saliva by observing the action on tooth structure of organic and therapeutic substances added to the saliva.

The procedure was as follows: Freshly extracted noncarious teeth were thoroughly cleaned. The whole tooth structure, devoid of pulp tissue, was used. These experiments were repeated later, with pure enamel and pure dentin fragments. Enamel samples showed smaller losses, but the results were parallel. Care was taken to prevent excessive dehydration or putrefaction. The teeth were broken into small pieces (approximately the size of a grain of wheat), carefully weighed and placed in a 50-cc. bottle containing only enough distilled water to prevent drying. Each bottle contained tooth structure weighing approximately 700 mg. (twenty pieces).

The bottle and contents were sterilized in an autoclave. Ten cubic centimeters of saliva was obtained from each of 100 subjects having from ten to twenty active carious lesions. The entire volume of specimen saliva was mixed together in order to obtain an average saliva the content of which is apparently conducive to dental caries. This mixture was called

2. Black, G. V.: Operative Dentistry, Vol. 1. Ed. 7. Chicago: Medico-Dental Publishing Co., 1936, p. 323.

susceptible saliva. In like manner, a 10-cc. specimen was obtained from each of 100 caries-free subjects and mixed as described above. This was then labeled immune saliva. Male subjects from 17 to 35 years of age were used. The saliva was collected at various hours of the day. Specimens were taken each hour from 8 a.m. to 4 p.m.

The tooth fragments previously prepared were again weighed and placed in separate specimen bottles, one group containing immune saliva and another susceptible saliva. The contents were then fortified with substances to be tested and were placed in an incubator. Twenty-four hours later, the tooth fragments were removed by sterile means for the initial weighing, and they were weighed twice weekly thereafter for a period of three months. The loss of weight of tooth structure represents a condition similar to common active caries. The dentin became leathery and reacted similarly to carious dentin from the standpoint of appearance and instrumentation after three weeks in the salivary sugar solutions. The pH of these solutions was 4.5. The enamel showed little change in structure or loss in weight. These conditions are similar to common caries and show a definite contrast to acid demineralization as observed by Frisbie *et al.*[3] and to other reactions of tooth structure in acid solutions.

Among the various organic compounds added to the saliva, immune or susceptible, were: (1) parotid extract, obtained from caries-free animals; (2) egg-sack extract, for calcium deposition; (3) thyroid extract, for metabolic value; (4) pituitary extract (anterior lobe), for its bone growth effect; (5) parathyroid extract, for calcium and phosphorus metabolism; (6) corpus luteum, ovarin and testicular extracts, because of dental disturbances manifest at puberty and during pregnancy and the menopause; (7) insulin, for sugar digestion; (8) saliva passed through a Berkefeld filter, for removal of certain bacteria; (9) bone flour, a calcium-phosphorus and fluoride mixture; (10) sulfonamides, for antibacterial activity; (11) penicillin, for antibacterial effect, and (12) sugar, because of its accepted stimulative action in regard to caries.

The first day's observation of these experiments revealed surprising results. It was noted that the addition of the various germicidal agents generally caused an increase in the solvent action of the saliva from the caries-susceptible. The greatest loss in weight of tooth structure in solutions containing organic or therapeutic agents occurred in the saliva to which sulfanilamide had been added. This solution showed a loss in weight of 25.74 per cent of the tooth structure submitted to its action. Of all substances tested, the greatest loss of tooth structure occurred in the solution of susceptible saliva to which 5 per cent sugar had been added. Here, a striking loss of 30.92 per cent was apparent in twenty-one days. This may indicate that sugar aids destructive organisms by its nutritional support. The saliva containing penicillin showed less solvent action than any other solution to which reagents had been added. The loss of structure here was but 1.77 per cent in twenty-one days. With the salivary solutions to which endocrine substances were added, results were not significant.

The saliva obtained from caries-susceptible persons and to which no reagent had been added showed, surprisingly, the slightest solvent action. The tooth structure submitted to its activity lost only 1.21 per cent. It was expected that the solvent action of this saliva would be high. Conversely, with the saliva

3. Frisbie, H. E.; Nuckolls, J., and Saunders, J. B. de M.: Distribution of Organic Matrix of Enamel in Human Tooth and Its Relation to Histopathology of Caries. J. Am. Coll. Dentists 11:243, September 1944.

Fig. 1.—Left: Inoculation with dilute saliva from caries-susceptible persons. Right: Inoculation with dilute saliva from caries-free persons.

from caries-free persons, there was a loss in weight of 2.11 per cent of tooth structure. This represented nearly twice the amount lost in the saliva obtained from the caries-susceptible. Thus, these results proved to be directly opposed to the expected outcome.

A rather limited bacterial study of the saliva used in the foregoing experiments proved interesting. Blood-agar petri dishes inoculated with dilute salivary specimens showed the presence of a nonfilterable hemolytic organism in numbers inversely proportionate to the amount of tooth structure dissolved in the respective solutions. (Figs. 1 and 2.)

Tooth structure submitted to the action of these specimens showed the following losses in weight:

1. Saliva obtained from caries-susceptible persons showing 8,000 hemolytic organisms per cubic centimeter, 1.21 per cent.

2. Saliva obtained from caries-free subjects showing 600 hemolytic organisms per cubic centimeter, 2.11 per cent.

3. Saliva from caries-susceptible subjects which has been passed through a Berkefeld filter and which shows no hemolytic organisms, 2.44 per cent.

These bacterial counts represent an average count taken from the saliva of more than 200 men of their respective groups. Specimens were obtained every hour from 8 a.m. to 4 p.m. The percentage loss in weight represents an average obtained by the repetition of this experiment more than twenty-five times.

This result suggests the possibility that

Fig. 2.—Inoculation with dilute saliva from caries-susceptible persons after passage of saliva through Berkefeld filter.

nature has provided a defensive organism in caries-susceptible mouths which acts in a manner similar to that of defensive factors present in other parts of the body. The number of those organisms present in the saliva can be compared with the number of leukocytes present in the blood stream; i.e., these defensive organisms, under natural conditions, increase in caries-susceptible saliva just as leukocytes increase in the blood stream in the presence of certain infections. It is assumed that these organisms are not found in predominant numbers in the saliva of caries-free persons because their presence is not necessary.

Jay[4] has suggested the possibility of a bacteriologic antagonist in caries control. An attempt to identify this organism was not successful in our laboratory. To secure expert assistance, cultures were sent to the U. S. Naval Hospital, San Diego, Calif., and to the Naval Medical Center, Bethesda, Md. Neither station met with any greater success, but, to demonstrate the thoroughness of the effort, the following report of the Naval Medical Center is presented:

A. Staining characteristics and microscopic morphology.

1. Gram stains showed the organism to be both gram negative and gram positive, but predominantly negative in old cultures.

2. Viewed microscopically, the organisms had the general structure of the streptobacillus, typical rod forms joined in chains and about 0.8 microns in width and 9.0 microns in length.

B. Cultural characteristics, from inoculation with organisms cultured in tryptose broth.

1. The optimum growth temperature was 37 C.

2. Definite liquefaction of gelatin was produced.

3. No hydrogen sulfide was produced on Kleigler's iron medium—a differentiation characteristic.

4. There was no coagulation in litmus milk and only a slight color change was noted, indicating that the organism was only slightly acid forming.

5. Dextrose, sucrose, maltose and mannite were fermented, with no formation of gas.

6. Lactose, xylose, dulcite and salicin were not fermented.

7. Hemolysis was not produced on a blood-agar plate, nor were there green colonies, the colonies present being slightly gray and non-hemolytic. [Observation was apparently made on a 24-hour culture. Hemolysis does occur after a forty-hour incubation period.]

8. With a culture shipped from San Diego, the organism, if spore bearing, would produce spores. There was no evidence of spore formation; i.e., it was non-spore bearing.

9. Only a scant growth was produced on Kulp's tomato-peptone-agar, an acid medium (pH 5.0).

10. No color change was produced in Snyder's bromcresol green-dextrose medium (pH 5.0).

C. Possible classifications and identification.

1. *Streptobacterium Orla-Jensen (Streptobacillus lebenis [Lactobacillus caucasicus])*[5] cannot be classified because the organism does not produce acid.

2. The streptobacillus of Rust and Khoury[6] identified as the Lactobacillus of Beijerinck, genus IV of the family IX Lactobacteriaceae,[7] cannot be classified because it does not produce acid from carbohydrates.

3. *Lactobacillus casei,* subgenus 9 of lactobacilli,[8] a short or long chain of short or long rod-shaped organisms, cannot be classified because it will not ferment lactose and salicin.

4. *Lactobacillus plantarum,* subgenus 10 of

4. Jay, Philip: Nutrition and Dental Caries. J. New Jersey D. Soc. 15:20, January 1944.

5. Mem. d. Acad. Sc. et Lett. de Denmark, Sec. Sc. 8, Ser. 5, No. 2, 1919.

6. Ann. Inst. Pasteur 16:70, 1902.

7. Orla-Jensen, S.: J. Bacteriol. 6:271, May 1921. Streptobacteriaceae. Bergey, D. H., et al.: Manual of Determinative Bacteriology. Ed. 5. Baltimore: Williams & Wilkins Company, 1938, p. 71.

8. Streptobacterium Casei-Orla-Jensen; Lactic Acid Bacteria. Holland J. Bacteriol. 5:223, 1920.

lactobacilli,[9] which may occur singly or in short chains with rounded ends, cannot be classified because it acts to cause liquefaction of gelatin and does not ferment lactose and salicin.

5. *Lactobacillus leichmannii,* subgenus 11 of lactobacilli,[10] which may occur singly and in short chains with two or more deeply staining granules, cannot be classified because it acts to cause liquefaction of gelatin.

6. The erysipelothrix of Rosenbach, genus 11 of the family 11 Actinomycetaceae of Buchanan,[11] rod-shaped organisms showing filaments and branching and characteristic granules and occurring singly and in chains, cannot be classified because its action causes liquefaction of gelatin and does not ferment lactose nor produce hydrogen sulfide. It is also apparently a saprophyte and not a parasitic form.

7. *Streptobacillus pseudotuberculosis rodentium (Bacillus pseudotuberculosis),* genus 7 of the family VIII Parvobacteriaceae of Rahm,[12] rod-shaped organisms occurring in groups or short chains, cannot be classified because its action produces liquefaction of gelatin and does not ferment xylose and salicin and will not produce hydrogen sulfide.

8. *Leptotrichia buccalis,* genus 1 of the family 11 Actinomycetaceae of Buchanan,[13] rods occurring in chains, often with partition walls, cannot be classified because its action causes complete liquefaction and not liquefaction cateriform, and also is not a parasitic form, but apparently a saprophyte.

D. Conclusions.

1. The organism submitted cannot be definitely classified into any definite genus or family of known micro-organism.[14]

2. An hypothesis may be voiced that the organism is one of the heterogeneous forms of saprophyte.

3. If further investigation is warranted, it is suggested that three or four more strains of the organism be submitted for parallel cultures of similar characteristics.

4. A bibliographic index is enclosed with this report in substantiation of the bacteriologic evidence submitted.[15]

This unidentified organism was isolated in pure culture, from which a heavy aqueous suspension of a 24-hour culture was made, which was added to a solution of saliva and sugar. Weighed tooth fragments were subjected to the action of this mixture. For twenty-four hours, a definite inhibitory effect was noted (0.52 per cent loss). The inhibitive effect was not prolonged, since a 4.67 per cent loss resulted in seventy-two hours and a 9.43 per cent loss in twenty-one days. These figures represent the average loss in weight obtained in repetition of the experiment at least twenty-five times.

Part 1: Inhibitory Factors Observed

At the conclusion of this simple test and a careful study of the graph in Figure 3, it occurred to us, notwithstanding all previously known findings, that oral secretions do contain a definite group of bacteria which act in antipathic symbiosis with dentally destructive organisms also present in the saliva, and that the disintegration of tooth structure can be retarded by the addition of certain organisms the function of which is apparently inhibitory to destructive processes. The disintegration of tooth structure can be hastened by:

1. The addition to saliva of a substance which kills or renders inactive the

9. Streptobacterium Plantarum Orla-Jensen, Lactic Acid Bacteria. Bergey, D. H., et al.: Manual of Determinative Bacteriology. Ed. 1, 1923, p. 250.

10. Bacillus Leichmannii I Henneberg. Ztschr. f. Spiritusindus. 26:22, 1903; Centralbl. f. Bakteriol. Pt. 2, 11:136, 1903; Bergey, D. H., et al.: Manual of Determinative Bacteriology. Ed. 2, 1925, p. 180.

11. Ztschr. F. Hyg. 63:367, 1909.

12. Bacillus Pseudotuberculosis Rodentium Schultze. M. Res. Coun. Sys. Bacteriol. 4:453, 1929. Topley, W. W. C., and Wilson, G. S.: Principles of Bacteriology and Immuniology. Ed. 1. London: Edward Arnold & Co. Vol. 2, 1931, p. 825.

13. Leptothrix Innominata Chester. Bergey, D. H., et al.: Manual of Determinative Bacteriology. 1901, p. 371.

14. Bergey, D. H., et al.: Manual of Determinative Bacteriology. Ed. 5, 1939, p. 39.

15. Murphey, P. J.: Unpublished data.

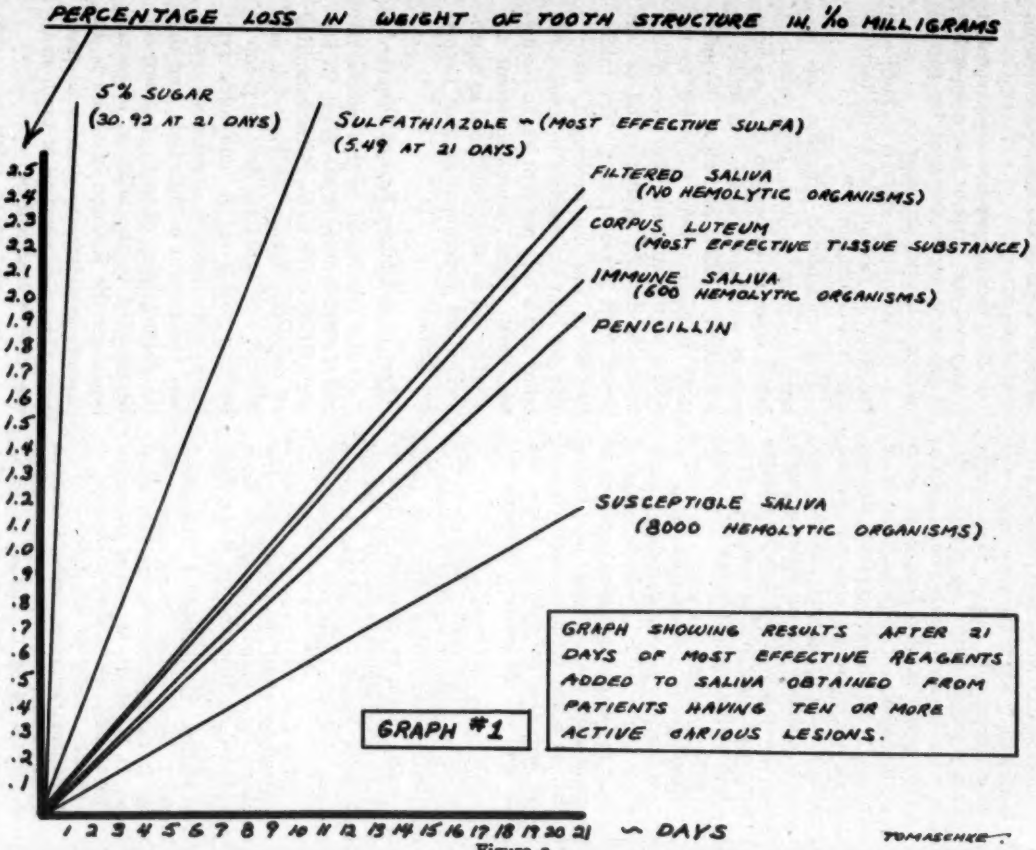

PERCENTAGE LOSS IN WEIGHT OF TOOTH STRUCTURE IN 1/10 MILLIGRAMS
5% SUGAR
(30.92 AT 21 DAYS)
SULFATHIAZOLE ~ (MOST EFFECTIVE SULFA)
(5.49 AT 21 DAYS)
FILTERED SALIVA
(NO HEMOLYTIC ORGANISMS)
CORPUS LUTEUM
(MOST EFFECTIVE TISSUE SUBSTANCE)
IMMUNE SALIVA
(600 HEMOLYTIC ORGANISMS)
PENICILLIN
SUSCEPTIBLE SALIVA
(8000 HEMOLYTIC ORGANISMS)
2.5 2.4 2.3 2.2 2.1 2.0 1.9 1.8 1.7 1.6 1.5 1.4 1.3 1.2 1.1 1.0 .9 .8 .7 .6 .5 .4 .3 .2 .1
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 ~ DAYS
GRAPH #1
GRAPH SHOWING RESULTS AFTER 21 DAYS OF MOST EFFECTIVE REAGENTS ADDED TO SALIVA OBTAINED FROM PATIENTS HAVING TEN OR MORE ACTIVE CARIOUS LESIONS.
TOMASCHKE

Figure 3.

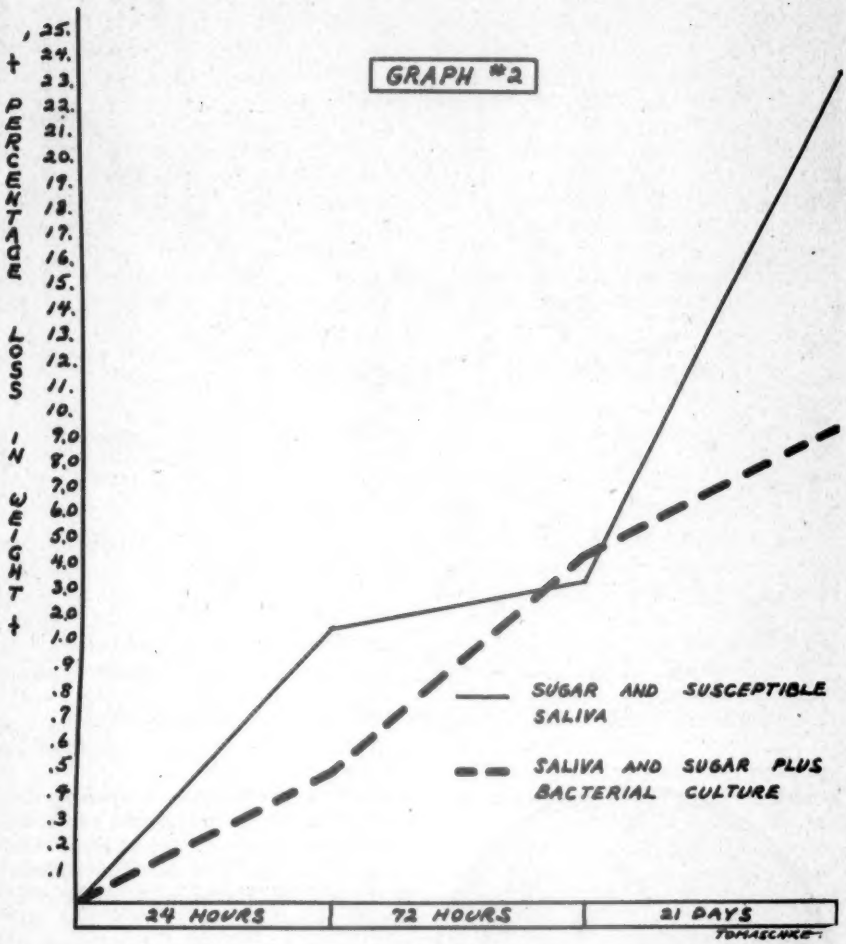


Figure 4.

protective organisms present in the saliva and which, at the same time, has little or no harmful action on destructive organisms.

2. The addition of a satisfactory nutrient, such as sugar, which aids the productive organisms to such a degree that the inhibitory organisms are outnumbered or rendered inactive. It may even be found that the destructive organisms, when present in predominant numbers, completely destroy and are nourished by the protective organisms in much the same manner as leukocytes are destroyed by harmful bacteria in phagocytosis.

For the sake of clarity: It is believed that two specific types or groups of bacteria or fungi are contributory to the production and inhibition of dental decay. It is not to be concluded that bacteria alone are the direct cause of decay. Rather it is supposed that they work in

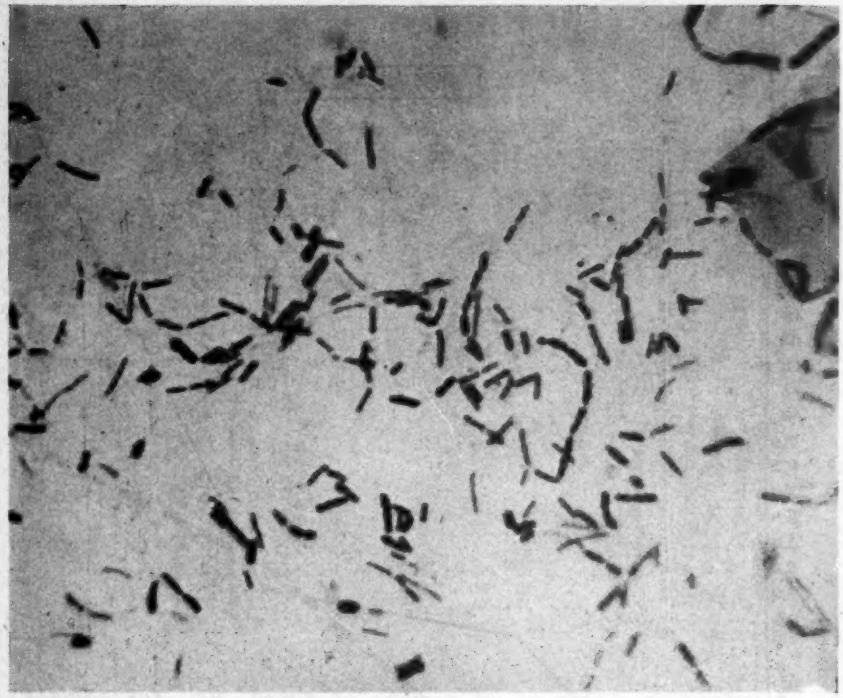


Fig 5.—Morphologic characteristics of unidentified organism described in text.

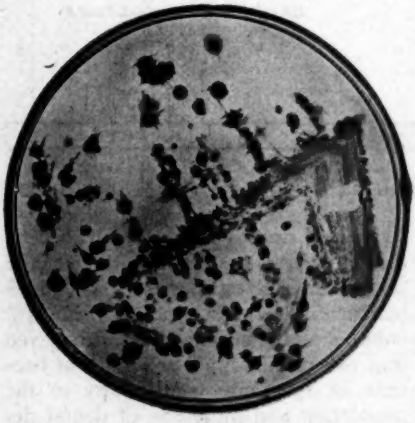


Fig. 6.—Cultural characteristics of streptobacillus described in text, as shown on nutrient agar medium.

a chain of constituents, comprising the positive factors in the actual condition found in the caries-free or in those with rampant caries. The bacterial content of the oral cavity may be, in either case, directly governed by hygiene and diet and by physical, emotional and environmental conditions.

In the foregoing hypothesis, may it be presumed that destruction of the protective organisms will influence the chain of conditions contributing to rampant caries?

Because of the prominence that sodium fluoride and penicillin hold in related medicodental literature, experiments were conducted along the lines previously described. Penicillin, 25,000 Oxford units, was added to 12 cc. of sus-

ceptible saliva which contained weighed tooth fragments. Likewise, sodium fluoride in a ratio of 1 to 500 was added to 12 cc. of susceptible saliva and weighed tooth fragments. The changes in weight are noted in the accompanying table.

The sugar was included in the table for comparison. The results obtained from using sodium fluoride were in agreement with those of a similar experiment conducted by Fosdick and Hansen in 1936,[16] in which no dissolution of enamel was noted when it was shaken with saliva preserved with sodium fluoride.

Changes in Weight of Tooth Fragments

	24 Hours	48 Hours	96 Hours
Sodium fluoride	1.10% gain	0.61% gain	0.93% gain
Penicillin	0.09% loss	0.43% loss	0.82% loss
Sugar (sucrose)	1.65% loss	2.66% loss	3.85% loss

The possible toxic effect of the presence of fluorides cannot be overlooked, as was recognized by Dean in 1943[17]:

> The possibility of cumulative toxic effects of a nondental nature at even a concentration of 1 ppm F cannot be entirely ruled out on the basis of evidence already presented. This possibility, however, seems rather remote as far as this country is concerned for it is well known that in the United States people have been using, for as long as forty years, waters whose fluoride concentrations were considerably higher and no known pathological effects, other than on the teeth, have been reported.

It is to be noted that the waters referred to by Dean were natural waters, not fortified by the addition of fluorides at the distribution center. A fatal dose of sodium fluoride is between 5 and 10 gm.[18] No doubt, much smaller dosages will have toxic systemic effects. The possible cumulative effect is not completely controlled when a substance is added to the water supply, for the amount of that substance retained is in direct proportion to the amount of water consumed per person.

The protective action of the unnamed streptobacillus on tooth structure is not so marked as the action of the sodium fluoride solution. However, the bacteria are nontoxic and nonpathogenic.

Three-and-one-half pound rabbits were inoculated with 1,000,000,000 of these unnamed living organisms from a 48-hour culture. No toxic effects were noted. It is realized that the effect upon human beings may be different, but it is not expected to be more toxic since the organism is a normal inhabitant of many human mouths.

The most effective method of utilizing these streptobacilli has not been determined, and probably many months of experimental work will be necessary to develop the product. The organic origin and experimental work to date reflect favorably on the success of the undertaking.

Part 2: Productive Factors Observed

Further observations demonstrate the plausibility of the presence of certain factors in dental caries, or necessary links in a complicated chain of chemical and bacterial activity, the end-products of which are dentally destructive substances.

Since tooth structure submitted to the action of the saliva and sugar solution

16. Fosdick, L. S., and Hansen, H. L.: Theoretical Consideration of Carbohydrate Degradation in Relation to Dental Caries. J.A.D.A. 23:401, March 1936.

17. Dean, H. T.: Domestic Water and Dental Caries. J. Am. Water Works A. 35: September 1943.

18. Wood, H. C., and La Wall, C. H.: U. S. Dispensatory. Ed. 22. Philadelphia: J. B. Lippincott Company, 1937, p. 1937.

shows the greatest loss in weight, it was decided to prepare a bacterial culture medium composed of a saturated solution of sucrose and 2 per cent agar, in an effort to obtain dentally destructive organisms. This medium was prepared and inoculated with saliva obtained from subjects having rampant caries.

After seventy-two hours' incubation, no growth of organisms was apparent. After ninety-six hours, a number of small round white colonies appeared. The entire growth was removed, and subculture revealed the presence of no other organism.

Microscopic examination revealed the growth to be a form of yeast. Weighed tooth structure inoculated with heavy aqueous suspensions of this yeast in pure culture and sucrose showed a loss in weight of 2.11 per cent in twenty-four hours and of 5.53 per cent in fourteen days. These statements represent an average obtained from repeated experimentation and supported earlier findings.[19]

A brief review of organic chemistry recalls that various sugars, when subjected to the action of yeast, produce alcohol and carbon dioxide:

$$C_6H_{12}O_6 + 2CO_2 \rightarrow 2C_2H_5OH$$

"Sucrose ($C_{12}H_{22}O_{11}$) in solution is decomposed by yeast into alcohol and carbon dioxide just as is grape sugar, only more slowly."[20] Alcohol is then partially oxidized into organic acids by bacterial action. Oral secretions may contain bacteria necessary for the formation of lactic acid ($CH_3—CHOH—COOH$) by lactobacilli and acetic acid (CH_3COOH) by *Bacillus aceti*. Carbonic acid is also formed by the liberated carbon dioxide and water ($CO_2 + H_2O \rightarrow H_2CO_3$). Although a weak acid, carbonic acid is active in converting insoluble calcium compounds into soluble substances—$H_2CO_3 + CaCO_3 \rightarrow H_2 + Ca + Ca(HCO_3)_2$.[21]

The formation of other acids has been recognized by Fosdick and other men working in this field, but our observations relative to the action of yeasts on carbohydrates limit our discussion at present to the three acids named above. The physiologic action of ptyalin on starches produces sugars, which, in turn, are oxidized into harmful acids.[20] These observations are not presented as original, for it was recognized as early as 1887 by Miller[22] and Black[23] that dental decay might be caused by the formation of lactic acid in the mouth as a result of carbohydrate fermentation. In the book "Dental Caries," second edition, 1941, published by the American Dental Association, yeast was associated with the production of dental caries by only five researchers (Atkins, Blayney, Florestano, Hansen and Hatton). The presence of yeast has been given but little consideration as a necessary link in the chain of conversion of sugars into dentally destructive acids. A new trend of thought presents the possibility of eliminating yeast from the chain of factors in dental decay, thereby interrupting the process and preventing completion of the chain necessary for the development of substances harmful to tooth structure.

Products inhibitory to the growth of yeasts and the formation of their enzymes are being studied at present.

Tests of saliva from the caries-susceptible to which sucrose had been added and which was incubated for forty-eight

19. Scrivener, C. A.: Dental Caries, Our Challenger. D. Items Int. 66: 1068, November 1944.

20. Kendall, James: Smith's College Chemistry. New York: Century Co., 1923, pp. 532-544.

21. Porter, C. W., and Stewart, T. D.: Organic Chemistry. Boston: Ginn and Co., 1943, pp. 142-267.

22. Miller, W. D.: Die Mikro-organismen der Munderhohle. Leipsic: George Theime, 1892.

23. Black, G. V.: Tr. Illinois D. Soc. 1886, p. 180.

hours showed a pH of 4.5. Although this acid formation is slow, it has been determined that when sugar is placed in a carious tooth or in a susceptible area of the oral cavity, the pH may drop to 4.0 in three minutes.[24] This is sufficiently low to cause demineralization of tooth structure.

Summary

Attention is called to certain possible factors in dental decay which heretofore have been given little consideration:

1. The use of mouth washes and dentifrices may be instrumental in promoting dental decay. These products may frequently destroy the delicate organisms that nature has provided as part of a defensive system against dentally destructive processes.

2. Certain strains of yeast are major contributors to the complicated chemical process involved in dental caries.

3. The presence of carbohydrates (especially sugars) provides a nutrient and creates a favorable host for certain dentally destructive organisms, most prominent of which are *Lactobacillus acidophilus* and yeast. If prolific growth of these organisms is induced, the protective bacteria are rendered ineffective and frequently are completely eliminated.

Attention is primarily directed to the caries-inhibitive action of a certain strain of unnamed nonpathogenic, nontoxic streptobacilli. To employ a product developed from these organisms, nature's own biologic defenses are utilized as a protection against a pathologic condition developed by man's own civilization. This provides a highly desirable approach toward elimination of any diseased condition and it is believed that acute caries will yield to this new, effective form of treatment.

THE BIOCHEMISTRY OF ORAL CALCULUS. II. THE PRESENCE OF CARBONIC ANHYDRASE IN HUMAN SALIVA*

Gustav William Rapp, Ph.D., Chicago, Ill.

In the first paper of this series,[1] a discussion of some conditions predisposing to oral calculus deposition was presented. It was shown that because of differences in calcium and phosphate concentration, calculus-positive saliva has a greater tendency to precipitate calcium-phosphate salts than has calculus-free saliva.

The significant point was raised then that a moderate amount of alkalinization of saliva with a tendency to calculus formation could cause sufficient oversaturation of calcium-phosphate salts in the saliva to facilitate precipitate formation. If we make the logical assumption that the elements of duct saliva are in equilibrium, we must realize that, within a brief time span, the normal saliva becomes slightly alkaline, as indicated by the difference between the measured pH and the "critical pH" (or equilibrium pH) of the saliva studied.

Alkalinization of saliva is probably the first step in the complex procedure

24. Fosdick, L. S.: Etiology and Control of Dental Caries. J.A.D.A. 29:2132, December 1, 1942.

*From the Department of Research, Chicago College of Dental Surgery, Dental School of Loyola University.

1. Rapp, G. W.: The Biochemistry of Oral Calculus. I. Conditions Predisposing to Oral Calculus Deposition. J.A.D.A. 32:1368, November 1-December 1, 1945.

of calculus deposition. This alkalinization must occur rapidly and near the salivary ducts. Such rapid alkalinization can occur if carbon dioxide is quickly lost from the saliva, since there is dynamic equilibrium among the carbon dioxide, the hydroxyl ion and the bicarbonate ion. When the carbon dioxide of this equilibrium escapes from solution, the hydroxyl ions accumulate to lend alkalinity to the solution.

A rapid escape of carbon dioxide from saliva depends on at least two conditions: (a) differences in carbon dioxide tension of the freshly secreted saliva and of the air in contact with it and (b) the presence of an enzyme (catalyst) which aids in the conversion of bicarbonate into carbon dioxide.

The first condition, the difference in carbon dioxide tension, is not easily measured. Estimates have been made, and it is suggested that the carbon dioxide tension of saliva is about 60 mm. of mercury. The carbon dioxide tension of expired air is around 40 mm. of mercury. This difference in tension would cause a loss of carbon dioxide from the saliva to the air.

The second condition is important since the amount of carbon dioxide as such in the saliva is relatively small. The presence of the enzyme carbonic anhydrase catalyzes the reactions:

(a) $H_2CO_3 \rightleftarrows CO_2 + H_2O$;

or simplified to indicate the net results:

(b) $HCO_3^- \rightleftarrows CO_2 + OH^-$.

These are truly equilibrium reactions and the enzyme will catalyze them in either direction, in accordance with the relative concentration of the reacting entities. If carbon dioxide is evolved from the saliva, the presence of the enzyme catalyzes the reaction and shifts it to the right. This forms more carbon dioxide, which can also be evolved, and thus the chain of events continues.

It is to be noted that, concurrently with a liberation of carbon dioxide, there is an accumulation of hydroxyl ions (OH^-). These lend alkalinity to the medium. This increase in alkalinity changes the equilibrium conditions of the insoluble electrolytes so that the solubility products are exceeded and precipitation may occur.

Experimental Data

The presence of carbonic anhydrase can be demonstrated in one of two general ways. The method of Meldrum and Roughton[2] depends on the rate of carbon dioxide liberation when the enzyme acts upon a dilute solution of sodium bicarbonate. The method of Keilin and Mann[3] depends on the rate of conversion of sodium carbonate into sodium bicarbonate when carbon dioxide is bubbled into the system. The presence of the enzyme carbonic anhydrase increases this conversion rate. With the aid of these two methods, we may measure the rates of reaction of both the uptake and the liberation of carbon dioxide.

In this study, both methods were employed. In Roughton's method, the rates of carbon dioxide liberation were measured in the presence of active and deactivated enzyme preparations and the rates compared. In Keilin and Mann's method, the length of time required for the color change of phenol red was used as the measure of activity. The length of time for color change with active enzyme preparations was compared with that of inactive.

The enzyme preparation was made as follows: Ten milliliters of paraffin-stimulated saliva was collected in a tube chilled in an ice bath. This saliva was placed in a cellophane bag and dialyzed against distilled water for eighteen hours at 10 C. The dialysate was used as the

2. Meldrum, N. U., and Roughton, F. J. W.: J. Physiol. 80:113, December 5, 1933.

3. Keilin, D., and Mann, T.: Biochem. J. 34:1163, 1940.

Table 1.—Carbonic Anhydrase in Human Saliva

	Evolution of Carbon Dioxide*			
Saliva	No Inhibitor	Boiled	Potassium Thiocyanate	Sulfanilamide
M1	120	40	42	44
M2	80	38	40	39
M3	210	42	42	42
M4	190	40	40	43
M5	75	41	43	45
F1	60	37	37	41
F2	160	42	45	41
F3	220	39	40	46
F4	120	40	42	39
F5	90	43	46	45

*Rate of carbon dioxide evolution, in apparatus units.

enzyme preparation. Dialyzation is necessary because normal human saliva has a high buffer capacity, which interferes with the proper determination of the end points.

For the control procedures, the enzyme was inhibited in one of three ways: (a) boiling for five minutes, (b) addition of potassium thiocyanate and (c) addition of sulfanilamide. The addition of the latter two substances almost completely inhibits the activity of the enzyme.

The results of the experiments are summarized in the accompanying tables.

In Table 1, the numbers represent the rate of evolution of carbon dioxide. Hence, the larger the numerical value, the greater the carbonic anhydrase concentration. The rates of evolution in the case of the preparations inactivated by boiling or by the addition of 0.0001 molar potassium thiocyanate or sulfanilamide represent the rate of the uncatalyzed reaction.

In Table 2, the numbers represent the time in seconds needed to convert sufficient carbon dioxide into carbonic acid to change the pH of the solution a predetermined amount. In this case, the lower the numerical value the higher the rate of catalysis. In both tables, the differences among the three values for the inactivated preparations give an approximation of the errors involved in each determination.

Table 2.—Carbon Dioxide in Human Saliva

	Carbon Dioxide Uptake*			
Saliva	No Inhibitor	Boiled	Potassium Thiocyanate	Sulfanilamide
M6	86	180	180	176
M7	95	178	178	180
M8	62	182	180	180
M9	160	175	174	176
M10	97	190	181	184
F6	85	180	179	176
F7	125	179	174	174
F8	170	178	177	179
F9	180	182	178	184
F10	94	180	182	176

*Time in seconds to cause color change of indicator.

Conclusions

1. The presence of the enzyme carbonic anhydrase in human saliva has been demonstrated. The presence of this enzyme can speed the uptake as well as the liberation of carbon dioxide.

2. The presence of 0.0001 molar concentrations of potassium thiocyanate and sulfanilamide almost completely inhibits the action of this enzyme in saliva. The effect of potassium thiocyanate might be significant, since this substance is often found in human saliva.

3. It is suggested that the presence of carbonic anhydrase in human saliva can speed the removal of carbon dioxide from the saliva near the salivary ducts. This loss of carbon dioxide is accompanied by alkalinization of the saliva, which, in turn, can allow the precipitation of calcium salts of phosphate and carbonate onto the tooth surfaces. Such precipitation can give rise to deposits recognized as oral calculus.

Further work is in progress in this laboratory attempting to show an interrelationship between the coincidence of carbonic anhydrase and its inhibitors in human saliva and the presence or absence of oral calculus.

THE DEVELOPMENT OF THERMAL CONTROL

Chester J. Henschel, D.D.S., New York, N. Y.

The era of the dry steel bur is rapidly passing, and with it will go much pain and trauma and many prolonged tedious operations. There are men who decry the use of the bur and advise hand instrumentation. Many dentists have always employed chisels, hoes and hatchets, but most have found them relatively ineffective unless supplemented by burs. There are those who use burs almost exclusively, and they are wrong in this; but so are the teachers who unrealistically overemphasize the virtues of hand instrumentation. The latter rightly fear the frictional heat and trauma of the dry bur; but the steel bur is here to stay indefinitely and so should be used to best advantage.

There is always a good deal of friction in the use of burs, and it has been estimated that, at second speed (about 2,000 revolutions per minute), the surface of a bur of relatively small diameter, such as a No. 558, travels over vital dentin at the rate of 25 feet per minute.[1] The moisture clinging to the bur is boiled off rapidly until the instrument itself is dry. As the mucin and débris were packed into a hard mass between the steel cutting blades, the bur became virtually useless. The dentist probably failed to realize exactly what was happening and reacted to the condition in a very human way, applying more speed and pressure, with the result that the tooth was not cut, but was worn down a little. In any event, a cavity was made, and so a filling was placed.

Pain was probably a sequel to this treatment, and perhaps the pulp died and the tooth was lost. Because of many such incidents, dentists became wary of the moist bur. They found it advantageous to isolate the area of operation with absorbents or a rubber dam. Many also

1. Henschel, C. J.: Friction of Revolving Steel Burs. J.A.D.A. 31:895, July 1, 1944.

thought that the final answer was never to "drill and fill" on the same day.

The pulp and vital dentin are especially sensitive to thermal changes,[2] and even a healthy pulp may be endangered by thermal shock when made more vulnerable to temperature changes by metallic restorations. Dentin and enamel are excellent thermal insulators,[3] but metallic dental prostheses are good conductors. We are aware of the vicious cycle that can be started by thermal irritation, since the physical limitations of the dental pulp are such that recovery from inflammation is problematical. When thermal shock is added to injury from the heat of cavity preparation, the results frequently may be disastrous. Many dentists learned to prepare a cavity and place a dressing before inserting the filling. The results of this treatment were fairly satisfactory; but the bur came to be a much feared instrument.[4]

It is relevant to mention here that those of us who use "thermal control" do "drill and fill" in the same day. With or without anesthesia, more rapid work is possible, but, so far as I know, there has been no postoperative hypersensitivity, despite the fact that steel burs were used in a wet field, a procedure frowned upon by leading educators. The idea of a wet field is, of course, nothing new.

It should be noted that, for a long time, no first-class metal workers have consistently used high speed drills without some form of cooling or lubrication. The dentist, working with tooth structure, saliva and mucin, has, since 1874, tried to combat operative friction by designing heat preventing appliances, which often have been patented.

In an old advertisement, there appears the picture of a dental handpiece fitted with a sort of nozzle and water-filled rubber bulb and hose for cooling a revolving stone. This apparatus, designed by George T. Barker, dates back to 1874. In 1903, Holson patented a spraying device for the foot engine, which delivered room temperature water by gravity from an overhead tank, the flow regulated by a finger-operated spring valve on the handpiece.[5] A month later, Lyman announced a more elaborate affair for air or water with provision for heating by an alcohol lamp placed beneath a reservoir.[6] Lyman's machine did not depend on gravity, as the water or air that it delivered was pumped by the foot pedal of the treadle-type engine and was, therefore, mechanically synchronized with the handpiece. It is also noteworthy that, in his drawings, Lyman depicted a small round instrument in the handpiece, which might have been a bur, whereas the previous, and most of the subsequent, inventors showed and wrote of grinding stones.

In January 1904, Gantz was granted a patent on a sprayer that featured a curved nozzle to wet the outer surface of a disk or stone. Otherwise, his device resembled Holson's.[7] About four years later, Craig patented a dental tool moistener, which was no improvement over its predecessors.[8] A palm-held rubber bulb and removable nozzle for the hand-

2. Jeserich, P. H.: Factors Necessary to Minimize Thermal Changes in Tooth Structure from Operative Procedures. New York J. Dent. 5:275, December 1935.

3. Behan, R. J.: Cephalalgia; Oral Diagnosis and Treatment Planning. (Edited by S. C. Miller.) Philadelphia: P. Blakiston's Son & Co., 1936, p. 20.

4. Henschel, C. J.: Importance of Pain Control. New York J. Dent. 11:438, December 1941.

5. Holson, J. C.: Dental Spraying Apparatus. U. S. Patent No. 739,674, September 22, 1903.

6. Lyman, Isidor: Chip-Blower and Syringe Attachment for Dental Engines. U. S. Patent No. 743,529, November 10, 1903.

7. Gantz, H. H.: Sprayer for Dental Polishing and Grinding Instruments. U. S. Patent No. 749,899, January 19, 1904.

8. Craig, St. George: Dental Tool Moistener. U. S. Patent No. 902,942, November 3, 1908.

piece was patented by Dollin and Couper in 1910.[9] In 1911, Cunningham and Smith were awarded a patent on an attachment very similar to Lyman's, except for a reversion to gravity flow.[10]

While all the early workers were correct in their theories, their appliances were crude and impractical. The first man after Lyman to make a substantial contribution to the problem was Hofschneider, who, in 1923, patented an electrically operated water or air valve for spraying revolving dental stones. This was marketed under the name "Oratunda" and a few were sold.[11] Hofschneider's machine, which was electrically synchronized with the dental engine, delivered an adjustable amount of warm water. It was undoubtedly the best product up to its time, but was cumbersome and unnecessarily complicated, and therefore quite expensive. Until recently, there has been at least one in operation. In 1926, Hofschneider produced a simpler device, mechanically operated by the engine cord. This model, which is still sold,[12] was the simplest and commercially the most practical product yet invented in this field, but it had several disadvantages, including a return to supplying cold (room temperature) water to thermal sensitive dentin unless the tank was freshly filled with heated water. Although criticism may logically be leveled at the apparatus, Hofschneider's models were mechanically sound, and their further development was undoubtedly prevented only by illness of the inventor before the devices were fully marketed.

In 1927, while still a dental student, I became interested in preventing the pain encountered in sterilizing and drying the prepared cavity and learned to warm alcohol in a porcelain dish before application. After graduation, in 1929, I began warming all medicinals prior to use, but it was not until about 1936 that the experiments crystallized in the "temperature tolerance zone."[13] During this period, it was proved that most dental reagents, including the caustic silver nitrate and the volatile chloroform, could be applied to even hypersensitive dentin without pain if the temperature of the tooth was not lowered in the process.

Meanwhile, Leon Lieber and I had been hearing almost unbelievable stories of freedom from trauma, and speedy, relatively painless cavity preparation with the use of wet burs with Hofschneider's machine. Many of these reports came from Jack R. Kopely, of Pelham, who, in turn, had learned the technic from Jacob Novikoff. Lieber and I, and then Morris Gudwin, joined the ranks of wet bur enthusiasts, and gradually it was realized that this method of treatment succeeded for the same reason that warming of medicinals tended to prevent pain. In the latter case, pain was eliminated by warming the medicinals and cotton pliers to body temperature and thus not lowering the surface temperature of the dentin. In "drilling," pain was prevented by not increasing the surface temperature of the dentin. In short, if the dentin was stabilized at body temperature level, dental treatment became relatively comfortable.

It is difficult to date most of this work, as we were all busy and did not make many notes, but I recall questioning some of Jeserich's statements when he lectured in 1935 at the New York Academy of Medicine. His work did

9. Dollin, A. F., and Couper, E. W.: Dental-Spray Attachment for Handpieces. U. S. Patent No. 953,022, March 29, 1910.

10. Cunningham, R. E., and Smith, A. C.: Air and Water Attachment for Dental Engines. U. S. Patent No. 1,000,785, August 15, 1911.

11. Hofschneider, F. J.: Device for Dental Use. U. S. Patent No. 1,466,299, August 28, 1923.

12. Hofschneider, F. J.: Liquid Feeding Attachment for Dental Engines. U. S. Patent No. 1,592,854, July 20, 1926.

13. Henschel, C. J.: Pain Control by Cold Control. D. Digest 47:444, October 1941.

not differ perceptibly from what was generally accepted up to and including that time. Dentists believed that the temperature rise caused by the use of small steel burs was negligible even during abuse and that a dry bur cuts best and its use causes a minimum of pain. These misconceptions arose from the almost universal failure to realize that frictional heat as developed by the use of steel burs is focused upon a relatively small surface of dentin, and the total heat is usually harmless to the pulp after the inevitable diffusion through bur and tooth structure. Those who measured the temperature rises of revolving burs seemed concerned with trauma and pulp death and the gross temperature change of the pulp or a fairly large mass of dentin, and were thus misled in their determinations. By this time, our group had noted that use of the smallest burs, even in the gentlest fashion, caused less pain if the bur was laved by a constant stream of water to reduce friction. This clinical observation seemed important and resulted in further work on the "thermal tolerance zone," which was determined to be from 85 F. to 130 F. for average dentin in a vital tooth.

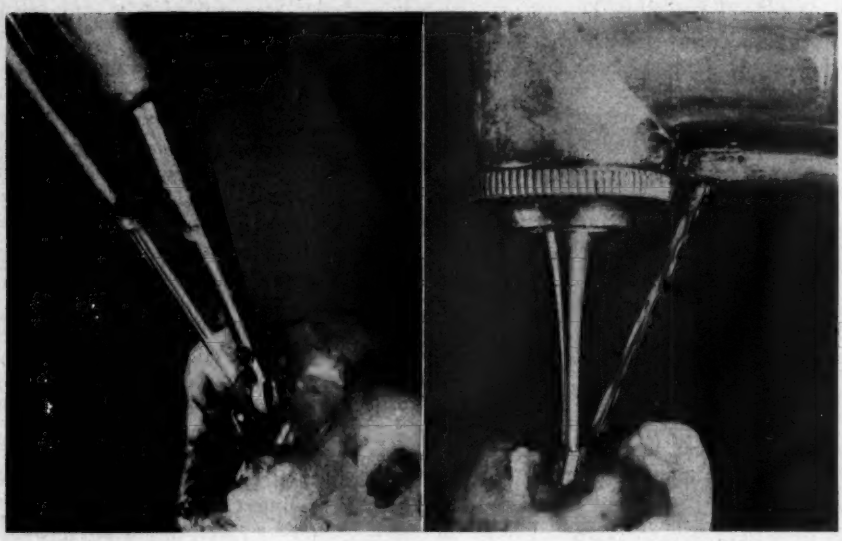
With this completed, it was apparent that the thermal rise even from the use of small burs must be more than 30 degrees if measured on the cut surface. After several years of experimentation, a simple method of doing this was found, and it was not surprising to find that the surface temperature of dentin, while being cut by a No. 558 bur, varied from about 166 to about 368 F., depending on whether 1 or 4 pounds pressure was used.[14] The experiments were performed at 2,000 revolutions per minute (second speed). Because of numerous factors in the experiment, these figures are minimal. They prove that the friction is certainly not negligible, as had been believed. It is true that many dry bur advocates had warned us regarding frictional heat, but our results scarcely agreed with the moderate figures published for even abusive treatment.

Abusive treatment, using high speed, more than several pounds pressure and the larger burs, results in many times the increase in temperature found during gentle operating and, in addition, generates so much caloric energy as to seriously endanger the pulp. While conventional dry, gentle operating causes frictional heat at the cut surface in excess of a 30-degree rise, beyond the limit of the thermal tolerance zone, and into the realm of pain, the total heat is probably harmless pathologically. Because of the limited effect of this type of frictional heat, it is almost irrelevant to differentiate between a surface temperature rise of 50 degrees and increases five or ten times that; therefore, the precise temperature is unimportant—maximum pain has been caused.

In following these principles to their logical conclusion, a physical law well recognized outside, but not within, the ranks of dentistry was evolved: The friction developed between two given surfaces is not dependent on the area of their contact, but only on the thrust or force between them. This explains how use of the smallest burs develops as much heat per dentin tubule as use of the larger varieties and often causes maximum pain by the intense focus of thermal irritation.

It is probable that the establishment of the thermal tolerance zone and the temperature determinations were distinct milestones in the history of "thermal control," since they explained why the stream of water was so effective in reducing pain, and were therefore useful in winning many converts from dental school teachings. By 1939, the work seemed sufficiently developed to permit public presentation and a series of study classes was arranged through the Tufts

14. Henschel, C. J.: Heat Impact of Revolving Instruments on Vital Dentin Tubules. J. D. Res. 22:323, August 1943.



Left: Water spray attached to contra-angle. Right: Water spray attached to handpiece.

College Study Club of New York. There began a long series of clinics and lectures before dental groups.

Meanwhile, Lieber, Kopely, Gudwin and I had become increasingly dissatisfied with the mechanical limitations and imperfections of the Hofschneider device. The tank was small, requiring constant refilling if used to any extent. The water was delivered cold (room temperature), and sometimes the synchronization failed. Lastly, and of the greatest practical importance, Hofschneider's design for the tubing, the clips or nozzles for the straight handpiece and especially those for the contra-angle was crude and unsanitary. Certain men operated under a constant stream of warm water from a syringe in the hands of a nurse, but this was impractical for many dentists, and not nearly so efficient as an automatic device. Therefore, an electrically synchronized apparatus was projected and, after hundreds of changes and dozens of models, the essential parts were presented at the Greater New York December Meeting in 1940. Instructions for assemblage were published in 1941.[15] It was not until the apparatus was completed that Hofschneider's early electrical device came to my attention—a device, which, although impractical, earns for its inventor a place of honor among the pioneers of thermal control.

Although it would be overoptimistic to consider the dry bur outmoded, "thermal control" is now in rather wide use and the technic is being used in many military and naval areas.

Because of war conditions, the apparatus has still to be somewhat improvised, but it is inexpensive and simple. Since any supply of warm water can be used, the easily held nozzle attachments constitute the most important innovation. After many different models had been made, with trials and errors, the cumbersome round metal tubing was developed into the flat "tape-tube" which closely conforms to any make of hand-

15. Henschel, C. J.: Pain Control by Heat Control. D. Digest 47:294, July 1941.

piece. Also, the nozzle, or teakettle-like water spout for directing the stream at the contra-angled bur, was eliminated, so there is now no projection to interfere with instrumentation.

Several recent devices should be mentioned. One, the Waropa, uses a complicated method of allowing liquid carbon dioxide to cool the bur by chilling the inside of the contra-angle or handpiece. This machine has some merit and usefulness, but is awkward to use, and is also bulky and expensive.[16] Another instrument, patented by Mulvany in 1939, reverts to air as the cooling agent, using the supply from the dental unit.[17] A New York dentist, Hyman Freedman, has also been trying to overcome operative friction, but with a different approach. Almost all of the previous apparatus was somehow connected or synchronized with the dental engine and the right, or working, hand. Freedman designed a saliva ejector which, on manipulation, sprays or squirts water. He also has perforated the dental mirror with a small tube for delivering a stream of water at the cavity. Thus, the left, or nonworking, hand may be usefully engaged.

The relative merit of all the foregoing apparatus is open to question, but it will be found that the temperature of water is more easily stabilized than that of air, for painless application, and its use does not involve the danger of desiccating dentin. Also water is unquestionably the best heat sponge that we know, and its use, properly applied, will prevent all thermal trauma even during the most violent abuse. H. A. Zander, of Boston, has been examining pulps, after cavity preparation (1) by orthodox methods, (2) with water and (3) with air, and his work may do much to crystallize opinion. In using water as a cooling or stabilizing medium, it must be constantly remembered that a moist bur clogs badly, but it will be found that when a preparation has been completed with a really wet bur bathed by a constant stream of warm water, both the bur and the prepared cavity are usually quite clean. It is not necessary to use an automatic device at all, but a good syringe in the hands of an assistant may suffice. Since, when once accustomed to the fluid stream, the operator and patient find dry "drilling" unsatisfactory, elimination of the uneconomic and relatively inaccurate manual application seems advisable.

In using water, several problems arose, but fortunately they were easily circumvented. Ejection is accomplished by any good saliva ejector and the clamp saliva ejector.[18] The effects of excessive wetting of the contra-angle are eliminated by 250 F. oil treatment,[19] which, coincidentally, sterilizes and lubricates.

On September 23, 1943, H. K. Warner, of the Australian College of Dentistry of the University of Melbourne, wrote of work done by H. E. Willis and himself: "The local temperature at the bur may under certain conditions exceed 250°C. (482° F.) as indicated by the temper colours developed thereon. The temperature in the pulp chamber approximately 2 mm. away may be as low as 50° to 60° C. (122°-140° F.)."[20] As their work was published in 1940, it seems that high frictional temperatures were suspected in Australia during the same period that our work developed in New York. The observation by Warner and Willis of the

16. Thomas, B. O. A.: Effectiveness of Chilled Burs in Eliminating Pain. J. D. Res. 20:361, August 1941.

17. Mulvany, M. J.: Dental Apparatus. U. S. Patent No. 2,231,393, February 11, 1941.

18. Henschel, C. J.: Clamp Saliva Ejector. D. Digest 49:350, August 1943.

19. Henschel, C. J.: Clinical and Laboratory Suggestions. III. Prolonging Usefulness of Contra-Angles. D. Digest 50:70, February 1944.

20. Willis, H. E., and Warner, H. K.: Heat Generated in Cavity Preparation. Austral. J. Dent. 44:62, February 1940.

temper colors developed on the steel blades of burs in clinical dentistry was timely, since our group no longer uses dry burs. It is probable that the work of the Australians is better confirmation of my findings than my own observations in drilling plastics and fusible metals.[1] In review, it is interesting that no one, as far as I could find, had deemed the specific heat of dentin or enamel worthy of determination.[14] Little work has been done in developing the bur, the most often used instrument and a virtual symbol of dentistry, but it is to be hoped that recent writings will add new impetus to research on revolving instruments. Meanwhile, an improvement may be expected from the chrome bur and diamond stones.

In this paper, an attempt has been made to tell the story of thermal control, and if credits have been omitted it is because I, like most, am only a part-time researcher. Mention may be made of the score who have been using water in their practices for many years. These men deserve thanks for their clinical corroboration, but it is regrettable that their enthusiasm was not more audible. Valuable technics should not be confined between the walls of operating rooms, and history will probably honor only those with the energy and conviction to crusade.

The tradition of the dry bur has been broken, but the bur itself remains an important item of our armamentarium. Let us use it to our own and our patients' advantage. "Thermal control" has been established, and may be applied in cavity preparation,[1, 14, 18] sterilization[13] of dentin and dental cementation.[21] These technics will do much to increase operative speed, safety and accuracy, and to minimize the need for anesthesia.

1235 Grand Concourse.

21. Henschel, C. J.: Effect of Mixing Surface Temperature upon Dental Cementation. J.A.D.A. 30:1583, October 1, 1943.

MORPHOLOGIC ALTERATION OF SMALLER ORAL TREPONEMES DURING AGING OF CULTURES; EFFECT OF AGE ON VIABILITY OF SPIROCHETAL CULTURES

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The spirochetes have presented greater difficulty from a technical standpoint than most other oral bacteria in isolation and maintenance on artificial culture media. Recently, several technics and media have been suggested [1, 2, 3, 4] for the primary isolation and cultivation of the smaller oral treponemes.

One of the problems confronting investigators in this field has been the maintenance of large numbers of stock cultures because of the frequency with which they must be transferred to prevent loss of strains. Wichelhausen and Wichelhausen,[3] who recommend the transfer of oral spirochetes every two to three weeks to maintain these organisms, indicate that cultures may remain viable for from four to six weeks.

In this laboratory, numerous strains of the smaller oral treponemes have been isolated during experiments designed to cultivate the various members of the oral spirochetal flora of man. Some of these strains have been maintained in cultures from five to thirty-one months without being transferred to fresh media in the interim.

It is the purpose of this paper to report some observations on the morphologic alterations of the smaller oral treponemes during the aging of cultures and the effects of aging on the viability of these organisms.

Materials and Methods

The strains of smaller oral treponemes used in this study were isolated from cases of Vincent's infection by a technic previously described.[4] A semisolid medium which contained a 1.4 per cent agar concentration was employed. This medium, essentially a modification of Huntoon's "hormone" agar, was enriched at the time of use by the addition of 1 cc. of ascitic fluid and 1 cc. of a 1 per cent solution of neutralized cysteine hydrochloride to 9 cc. of the agar base.

All cultures of treponemes were isolated and maintained in jars rendered anaerobic with hydrogen catalyzed by electrically heated platinized asbestos coils, and incubated and maintained at 37 C.

Results

All of our laboratory strains of the smaller oral treponemes are routinely maintained from two to four months by methods previously designated, before being transferred. Prolonged maintenance on culture media has not resulted in undue prolongation of the lag phase

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1. Kast, C. C., and Kolmer, J. A.: Methods for Isolation and Cultivation of Treponemes, with Special Reference to Culture Media. Am. J. Syph., Gonor. & Ven. Dis. 24:671, November 1940.

2. Rosebury, Theodor, and Foley, Genevieve: Isolation and Pure Cultivation of Smaller Mouth Spirochetes by Improved Method. Proc. Soc. Exper. Biol. & Med. 47: 368, June 1941.

3. Wichelhausen, O. W., and Wichelhausen, R. H.: Cultivation and Isolation of Mouth Spirochetes. J. Bacteriol. 43:65, January 1942.

4. Hampp, E. G.: Method for Routine Isolation and Cultivation of Smaller Oral Treponemes. J.A.D.A. 30:1066, July 1, 1943.

in subsequent cultures. The first indication of visible growth becomes apparent on an average of thirty-six hours after inoculation of the media and, once initiated, progresses in an expeditious manner, reaching maximum growth in an average of five days. At this time, the visible growth extends outward from the central stab canal as a homogeneous opalescent haze, which is not sharply demarcated from the surrounding medium. Dark-field examination of spirochetes obtained from these cultures reveals organisms of typical structure as evidenced by their closely knit, symmetrically spaced spirals and delicately pointed ends.[5, 6] Motility is rapid and action rotating.

Associated with the aging of cultures, the characteristic homogeneous haziness of the spirochetal growth gradually changes until it presents a granular appearance and its opalescent whitish color, in most instances, gradually gives way to a pale yellowish cast. These gross visible changes are evident after about three months, becoming more pronounced with the progression of time.

Periodic dark-field examination of cultures of the smaller oral treponemes indicates that, about three weeks after inoculation, certain alterations in structure become apparent. During this period, the outstanding change is the appearance of long chains of spirochetes, which are not in evidence in appreciable numbers during the logarithmic phase of growth. These chains may be so long as to extend beyond the vision of a ×1,400 microscopic field. Many of these chains show small nodal points apparently representing areas at which the chain will divide transversely to form individual cells. There is a gradual transition from forms with closely knit, symmetrically spaced coils to forms with irregular spirals separated by areas which are straight. Motility also decreases at this time. Many of the spirochetes become granular. Associated with this change is the extension of one or more extremely fine coiled protoplasmic filaments from the terminal ends or from the sides of the cell. (Fig. 1.) At the same time, spirochetal granules, approximately 7 microns in diameter, become evident. These are either intimately associated with the cell bodies or found free in the media. Those granules attached to the spirochetes may occupy a middle, subterminal or terminal position in respect to the cell body and are usually external. They are rarely found within the somatic substance.

In the early stages of formation, the granules are uniformly round, having an appearance which is reminiscent of a nucleus of a tissue cell, with its characteristic chromatin network. Under dark-field illumination, they appear to have a bright white, highly refractile external membrane and a finer lacelike interior structure containing a small bright white, highly refractile body. Many of the

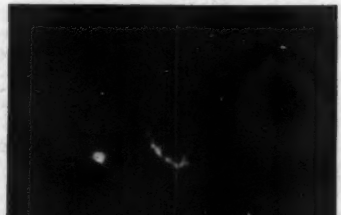

Fig. 1.—Dark-field photomicrograph of smaller oral treponeme prior to granule formation, exhibiting formation of two fine coiled protoplasmic filaments. (1455.)

5. Hampp, E. G.: Comparative Study of Dark-Field and Stained Smear Technics for Identification of Oral Spirochetes on Basis of Morphologic Characteristics. J.A.D.A. 32: 318, March 1945.

6. Hampp, E. G.: Vincent's Infection—A Wartime Disease. Observations on Oral Spirochetal Flora Present in Vincent's Infection. Am. J. Pub. Health 35:441, May 1945.

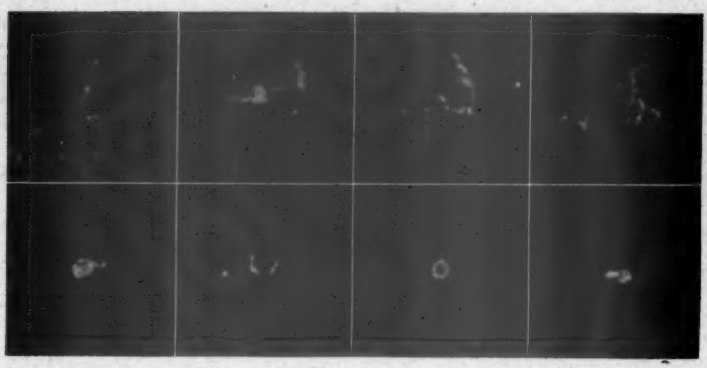

Fig. 2.—Composite of eight dark-field photomicrographs exhibiting examples of spirochetal granules. (1455.)

granules possess portions of a coiled treponeme extending from the external membrane. These are of various lengths, but do not exceed in length a single spirochete. Occasionally, two appendages may be connected to opposite poles of one granule. Figure 2 is a composite of several dark-field photomicrographs exhibiting examples of these structures. With aging of cultures, these granules become progressively smaller, condensing so that at about 7 months they appear as small condensed, highly refractile bodies approximately 3 to 4 microns in diameter.

These spirochetal bodies in the initial stages of formation do not react as do spores to the common bacterial stains. On the contrary, the granules, in the early stage, in which they resemble the nuclei of tissue cells, stain in a manner similar to that of a nucleus and consist of a membrane and a fine interlacing interior network. When overstained, these structures take on a deep, homogeneous color and the internal structure is obliterated. Spirochetal appendages attached to the spherules take the stain in approximately the same manner as normal spirochetes. In the later stages of granule formation, when the granules appear as condensed, highly refractile bodies under dark-field illumination, certain of them show varying intensities of coloration, when stained, while occasional spherules will not take the stain and remain colorless. This would seem to confirm our dark-field observations that these spherules are of various densities as revealed by the manner in which light is refracted.

In addition to the foregoing types of granules, there are found other bodies which appear to be formed by the rolling up of the spirochete. This is evidenced by the spiral outline of the external membrane of the sphere. (Fig. 2, upper right.) Mudd, Polevitzky and Anderson[7] have described similar forms in electron microscopic studies of spirochetes and refer to them as "end bodies." Whether this stage represents the initial step in the formation of the spirochetal granule is debatable. Warthin and Olsen[8] are of the opinion that such forms of *T. pallidum*, observed in focal aortic lesions in

7. Mudd, S.; Polevitzky, K., and Anderson, T. F.: Bacterial Morphology as Shown by Electron Microscope. J. Bacteriol. 46:15, July 1943.

8. Warthin, A. S., and Olsen, R. E.: Granular Transformation of Spirocheta Pallida in Aortic Focal Lesions. Am. J. Syph. 14:433, October 1930.

Viability of Smaller Oral Treponemes Maintained for from Five to Thirty-One Months on Veal Heart-"Hormone"-Agar at 37 C. Under Anaerobic Conditions

Strain	Age at Time of Transfer Months	Degree of Growth at Certain Hours After Inoculation of Fresh Media						
		24	48	72	96	120	144	168
N-54	5	*	*	*	*	*	++++	++++
N-60	5	*	*	*	*	*	++++	++++
N-59	5	*	*	*	*	*	++++	++++
N-14	6	0	0	+	++	+++	++++	++++
B.T.	6	0	0	+	+	++	++++	++++
N.F.	9	0	0	+	++	+++	++++	++++
M.J.	9	0	0	+	++	+++	++++	++++
G.M.	10	0	+	+	++	+++	++++	++++
F.M.	10	*	*	*	*	*	++++	++++
W.M.	10	*	*	*	*	*	++++	++++
Wave	11	*	*	*	*	*	++++	++++
J.B.	11	*	*	*	*	*	++++	++++
M.Pt.	11	*	*	*	*	*	++++	++++
N-O	12	*	*	*	*	*	++++	++++
N-1	12	*	*	*	*	*	++++	++++
N.N.	12	*	*	*	*	*	++++	++++
N.G.	13	*	*	*	*	*	++++	++++
J.H.	13	*	*	*	*	*	++++	++++
M.R.B.	13	*	*	*	*	*	++++	++++
C.T.**	14	0	0	0	+	++	+++	++++
N.G.**	14	0	0	+−†	++	++	++++	++++
M.R.B.**	14	0	0	+†	++	+++	++++	++++
J.H.**	17	0	0	0	+	++	++++	++++
J.H.**	19	0	0	0	+†	++	++++	++++
N.G.	19	0	0	+−	++†	+++	++++	++++
G.M.**	19	0	0	++	+++	++++	++++	++++
J.H.	21	*	*	*	*	*	++++	++++
N.G.**	22	0	0	+−†	++	++	++++	++++
N-1	22	0	0	+−	+	++	+++	++++
G.M.	22	0	0	0	+	++	++++	++++
J.H.	22	*	*	*	*	*	++++	++++
N-1	23	0	0	+	++	+++	++++	++++
G.M.	23	0	0	0	+	++	++++	++++
N.F.	24	0	0	+	++	+++	++++	++++
N-2	24	0	0	+	+	++	++++	++++
1-A	24	*	*	*	*	*	+++	++++
N-G	24	*	*	*	*	*	++++	++++
J.H.	31	0	0	+	++	+++	++++	++++

*Medium not examined at the hours indicated.

0, no growth.

+ to ++++, degree of growth.

**These cultures had been found on a previous occasion to survive from eight to fourteen months without being transferred.

†These cultures did not produce the characteristic spirochetal haze at this time, but formed a heavy condensed growth along the central stab canal. The characteristic haze was regained after several passages.

syphilis, represent the first step in the formation of spirochetal granules. This observation does not coincide with our findings on the oral spirochetes. By far the greatest number of granules observed in our studies have been found

in association with more or less typical spirochetal cells.

With progressive aging of cultures, proliferation of spirochetal granules continues until, at approximately 7 months, such cultures are found to be devoid of vegetative forms of organisms as far as can be determined by dark-field examination.

In order to check the viability of the smaller oral treponemes during the process of aging, forty-five cultures varying from 5 to 31 months of age were utilized. The total of forty-five cultures consists of the thirty-eight listed in the table plus those referred to in the two star footnote. Contained in this series of cultures were twenty-three individual strains of treponemes. All cultures were examined by dark-field illumination before subcultures were made. With the exception of strains N-54, N-60, N-59, N-14 and B.T., all the cultures, as far as could be visibly ascertained, contained only highly refractile granules. The former strains were found to contain occasional vegetative forms exhibiting granular degeneration, irregular spirals, blunted ends and loss of motility. After microscopic examination, the cultures were transferred to a veal heart-"hormone"-agar containing cysteine hydrochloride and ascitic fluid and incubated at 37 C. in anaerobic jars. At the end of seven days, all subcultures contained four plus growth as shown in the table. With the exception of the strains indicated, all strains produced a typical spirochetal haze extending outward into the surrounding medium. Those cultures that did not produce the spirochetal haze showed a heavy condensed colony-type growth along the stab canal with slight haziness in the immediate vicinity of growth. The ability of these strains to produce the characteristic spirochetal type of growth was regained after several passages.

Dark-field examinations of all cultures revealed in every instance numerous

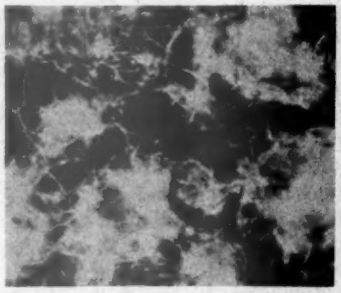

Fig. 3.—Dark-field photomicrograph of 6-day-old culture of smaller oral treponemes (strain J.H.), subcultured from a 31-month-old culture. (645.)

organisms with the typical morphologic characteristics previously described. No attempt was made to estimate the number of organisms per microscopic field because of the overwhelming number present and the characteristic clumping. These findings are adequately portrayed by Figure 3, a dark-field photomicrograph of a 6-day-old culture of strain J.H., which was subcultured from a 31-month-old culture.

Comment

In our experiments designed to isolate the various members of the oral spirochetal flora, numerous pure strains of the smaller oral treponemes were obtained. Observation of these cultures indicated that these organisms were extremely resistant and survived for long periods without being transferred. It is usually considered that the spirochetes are fastidious organisms as regards artificial cultivation and that frequent transfers are necessary to maintain stock cultures. Our findings do not agree with such an hypothesis. Observations of cultures of various ages were made and morphologic alterations during the process of aging were recorded.

One of the most constant findings in these experiments was the formation of spirochetal granules. The significance of

the role of these granules in the life cycle of the spirochetes has provoked conjectures as to the possibility of their being germinative units similar to spores. Certain investigators have indicated that this is a possibility.[7, 9, 10, 11, 12] Other investigators are undecided or hesitant in accepting this hypothesis.[8, 13, 14, 15]

The constant finding of granules in association with the various types of spirochetes lends a certain amount of significance to the possibility that they do not represent degenerative products or artefacts. They are definite and characteristic spherical bodies originating in association with the spirochetal cell and are of rather uniform structure and highly refractive, more so than the organisms from which they are derived. They are formed in an unorthodox position in relation to the cell body as compared with the endospores of bacteria, but are intimately connected with the cell body of the spirochete. From observation of our pure cultures of the oral spirochetes, it has been found that "granule production" seemingly represents a constant and rhythmic process in the life cycle of these organisms. These structures make their initial appearance at about 3 weeks and continue to increase in numbers until at about 7 months, when no vegetative forms can be discerned by dark-field examination.

Of the forty-five cultures of the oral spirochetes used in this study, only five strains of organisms, from 5 to 6 months old, were found to contain nonmotile, distorted vegetative forms on dark-field examination. All the others, as far as could be ascertained, contained only granules. Of this group of cultures, twelve varied in age from 21 to 31 months, and, when they were transferred to fresh media, produced characteristic growth in a short time. These findings seemingly indicate that the spirochetal granules may have a positive significance in the life cycle of the organisms.

Routine observations of spirochetal cultures indicate that, at about 3 weeks, long chains of organisms become evident which are not present in any appreciable number during the logarithmic phase of growth. There is a gradual transition from the typical closely knit, symmetrically spaced spirals to irregular coils. Associated with these changes is a decrease in motility. In addition, extremely fine coiled protoplasmic filaments become evident, extending out from the ends or sides of the cell body. At this period, spirochetal granules become apparent and are intimately associated with the spirochetes and seemingly represent a rhythmic process in the life cycle of the organisms. This process continues until all vegetative forms are replaced by these structures.

Forty-five cultures of the smaller oral treponemes were used in this study, varying from 5 to 31 months of age without having been transferred. When subsequent transfers were made to fresh media, growth was initiated in a characteristic manner without undue prolongation of the lag phase of growth.

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10. Noguchi, Hideyo: Spirochetes. Am. J. Syph. 1:261, April 1917.

11. Leishman, W. B.: Note on "Granule-Clumps" Found in Ornithodorus Moubata and Their Relation to Spirochetes of African Relapsing Fever (Tick Fever). Trop. Dis. Bull. 12:209, September 1918.

12. Balfour, A.: Infective Granule in Certain Protozoal Infections as Illustrated by Spirochaetosis of Sudanese Fowls. Brit. M. J. 1:752, April 1911.

13. Fantham, H. B.: Spirochaetes and Their Granular Phase. Brit. M. J. 1:409, March 1916.

14. Akatsu, S.: Influence of Carbohydrates on Cultivation of Spirochetes. J. Exper. Med. 25:375, March 1917.

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"GERODONTOLOGY": THE STUDY OF CHANGES IN ORAL TISSUES ASSOCIATED WITH AGING*

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The age of our population is constantly increasing. Fishbein[1] has said:

Out of 100,000 infants born in 1876 only 30,000 were expected to survive to the age of 65. Actually, however, 42,000 survived to the age of 65 because of the improvement in medical science, public health and general welfare. In 1850, only 2.6 per cent of our population were 65 years of age. In 1900, it was 4.1 per cent and in 1940, 6.8 per cent. In 1980, it is estimated that those who are 65 years of age or over will constitute almost 15 per cent of our total population.

Brunot writes, "The total population of New York City increased only 7.6 per cent in the decade following 1930, but the number of persons aged 65 and over increased 56.7 per cent."

Since more people are attaining old age today than 100 years ago, the health professions are evincing an increasing interest in the problems associated with aging. This interest in age changes has brought with it a new terminology relative to this special field: Geriatrics is the treatment of diseases of old age. Gerontology is the study of the biologic, physiologic and pathologic changes of senescence and senility. Gerontotherapy is the treatment of the aging process as an entity.

Gerontology

What has gerontology yielded in the way of specific knowledge? Let us consider some age changes in various components of the body.

The Cell.—Cowdry[2] (1943) says:

Some kinds of cells are evidently replaced thousands of times in the human life span, others hundreds of times, and still others not at all. . . . The long-lived ones (cells) which are not replaceable accumulate pigments, lipoids and various other relatively inactive materials, which suggest a diminution in physical vigor.

There is a gradual retardation of cell division, lessened capacity of cell growth, cellular atrophy, degeneration, increased cell pigmentation and fatty infiltration. The chemical nature of the cell may vary during aging with or without a concomitant morphologic change. The rate of aging varies with the type of cell, according to Stieglitz.[3]

True fatty degeneration occurs in the nerve cells of old people. Chemical age changes in muscle suggest a decrease in intracellular fluid and an increase in intercellular fluid. Age changes have not been recorded in smooth muscle, but progressive changes are well known in striated muscle—fatty infiltration and brown atrophy, with alterations in the thickness and cross-section contour of the fibers.

Intercellular Substance.—Inanimate parts, which do not consume oxygen, including various fibrous structures, fluids, the matrix of hard structures such as bone and cartilage and in fact all noncellular constituents (and erythrocytes), change with time. There is some shrinkage and condensation of intercellular

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1. Fishbein, Morris: Problems of Aging Population. Hygeia 20:813, November 1942.

2. Cowdry, E. V.: Factors in Aging. Sc. Month. 56:370, April 1943.

3. Stieglitz, E. J.: Pertinent Problems of Geriatric Medicine. Ann. Int. Med. 18:89, January 1943. Geriatrics in Wartime. M. Ann. Dist. of Columbia 12:19, January 1943.

substance, while in muscle there is increased intercellular fluid.

Tissues.—Blood vessels are hardened through deposition of fibrous tissue and calcium. As a result of a decreased flow of blood to the tissues in old age, there is an impairment of their nutrition and an increase in waste processes. In bones, the osteocytes and osteoblasts are affected, and the bone becomes more brittle and easily broken and heals slowly when broken. The bone marrow remains relatively normal in the aged. White connective tissue fibers, which possess great strength and hold the tissues together, can be formed throughout life, but replacement is inadequate because the old fibers are not removed in an orderly way, like dead cells, but tend to remain in the tissues.

The elasticity of the blood vessels, urinary bladder, skin and many tubular and other structures depends on the yellow elastic connective tissue. Even in advanced years, new elastic fibers are developed, but here too the mechanism of replacement is altogether inadequate. The most common age changes in skin are atrophy and degeneration. The ability of the skin to heal is greatly retarded, owing to decreased capacity for regeneration. Loss in elasticity seems to result from actual degeneration of the elastic connective tissue and perhaps partly from the decrease in subcutaneous fat. Decreased secretion of the sebaceous glands may be responsible for the dryness of the skin of the aged.

There are numerous ideas concerning the fundamental nature of the changes associated with aging. Davis[4] (1943) writes, "The problems of senescence will undoubtedly be solved when cellular chemistry can explain the involutionary changes that characterize it." He believes that heredity and one's constitution must not be overlooked as factors in age changes. In another paper[5] (1941), he says, "It seems, when all the evidence is assembled, that it has almost been proved that the involutionary changes of aging and the consequent degenerative diseases are due to the changes in cellular chemistry that result from various types of chronic suboptimal nutrition." Bogomolets[6] asserts that the age and health of the body are determined largely by the age changes and physiologic condition of its connective tissue, which interacts with other physiologic systems of the body.

There is no reason to assume that such age changes fail to occur in the tissues of the oral cavity. Although considerable work has been done in studying such changes in the oral tissues, much more remains for future study. I agree with Stieglitz[2] that "basic data concerning the biochemical, structural and physiologic changes which constitute normal aging are necessary for the differentiation of normal senescence from pathologic states," and "we need to know much, much more of just what aging is and does."

Gerodontology

Gardner[7] has suggested that the term geriodontia be applied to dental treatment of old people. Believing that the scientific study of the changes occurring in the dental tissues with age is becoming increasingly important, I suggest that the term gerodontology be applied to this special phase of dental research. The word gerodontology as defined in the title of this paper has the same

4. Davis, N. S., III: Factors Which May Influence Senescence. Ann. Int. Med. 18:81, January 1943.

5. Davis, N. S., III: Role of Nutrition in Treatment of Involuntary Processes of Aging. Tr. Am. Ther. Soc. 41:67, 1941.

6. Bogomolets, A. A.: Antireticular Cytotoxic Serum as Means of Pathogenetic Therapy. Am. Rev. Soviet Med. 1:101, December 1943.

7. Gardner, B. S.: Personal communication to author.

significance in the field of dentistry as the term gerontology has in medicine.

The remainder of this paper will deal with the present status of our knowledge as regards gerodontology. For convenience, the dental tissues will be discussed individually. In this respect, I have followed the outline of Robinson, Boling and Lischer.[8]

Enamel.—Attrition is the process producing wear of the enamel on its occlusal surface and at the proximal contact points. Although this causes little change in the enamel other than loss of bulk, the effects in dentin, pulp and parodontium are far-reaching. A number of investigators, including Box,[9] Keith,[10] Campbell,[11] Linghorne[12] and Begg,[13] consider attrition to be a normal aging mechanism and assign to it the definite function of preventing parodontal disease. Abrasion of the crowns of teeth alters the relation of mandible to maxilla, as well as the relation of the individual tooth to the alveolar process.

Bodecker and Lefkowitz[14] (1937, 1939) found that with maturity the permeability of dental tissues, including enamel, was greatly reduced. This decreased permeability is probably related to the desiccation that occurs in all tissues with age.

Erosion is characterized by loss of substance, beginning on the surface of the enamel and leaving a smooth, hard, polished surface. It is assumed to be the result of a physicochemical process. Erosion and cervical abrasion are much more common in older groups.

Dentin.—As a result of aging, a change occurs in the dentin, resulting in closure or blocking of the previously open lymph-filled dentinal tubules. Lefkowitz[15] (1942) has shown the extent of this metamorphosis at various ages. Kronfeld[16] writes:

> With advancing age, the vitality of the dentin is greatly decreased by sclerosis and obliteration of the tubules and by secondary dentin formation which cuts off the tubules from the pulp. Old dentin may become entirely insensitive and impermeable.

I have demonstrated,[17] clinically and histologically, that such metamorphosis in the dentin results in decreased sensitivity during cavity preparation. Orban[18] says, "With advancing age, the width of the dentinal tubules is said to become narrower." The size and form of the pulp chamber change as a result of

8. Robinson, H. B. G.; Boling, L. R., and Lischer, B. E., in Problems of Aging, Ed. 2, edited by E. V. Cowdry. Baltimore: Williams & Wilkins, 1942.

9. Box, H. K.: Significance of Wear in Physiology of Masticatory Apparatus. Oral Health 29:837, November 1939.

10. Keith, A.: Concerning Certain Structural Changes Which Are Taking Place in Our Jaws and Teeth. Brit. D. J. 45:1243, September 15, 1924.

11. Campbell, T. D.: Dentition and Palate of Australian Aboriginal. Adelaide: Hassell Press, 1925.

12. Linghorne, W. J.: New Theory of Nature's Plan for Human Dentition. Oral Health 28:525, December 1938.

13. Begg, P. R.: Progress Report on Observations on Attrition of Teeth in Relation to Pyorrhoea and Tooth Decay. Austral. J. Dent. 42:315, September 1, 1938.

14. Bodecker, C. F.: Variations in Lesions and Activity of Dental Caries. J. D. Res. 16:51, February 1937. Bodecker, C. F., and Lefkowitz, William: Concerning "Vitality" of Calcified Dental Tissues. J. D. Res. 16:463, December 1937. Lefkowitz, William, and Bodecker, C. F.: Concerning Vitality of Calcified Dental Tissues; Permeability of Enamel. J. D. Res. 17:453, December 1939.

15. Lefkowitz, W.: "Vitality" of Calcified Dental Tissues. V. Protective Metamorphosis of Dentin. J. D. Res. 21:423, October 1942.

16. Kronfeld, Rudolf: Dental Histology and Comparative Dental Anatomy. Philadelphia: Lea & Febiger, 1937.

17. Thomas, B. O. A.: Protective Metamorphosis of Dentin—Its Relationship to Pain. J.A.D.A. 31:459, April 1944.

18. Orban, Balint, quoted in Problems of Aging, by E. V. Cowdry. Baltimore: Williams & Wilkins, 1939, Chap. 11, p. 330.

the deposition of secondary dentin. Bodecker[19] (1925) describes several forms of secondary dentin, based on the function or etiology of each type. Secondary dentin is not always formed, owing to inactivity of the pulp for unknown reasons. The tubules in secondary dentin are sparse and irregular, owing to the fact that odontoblasts degenerate with advancing age or are destroyed by irritation, whereas the matrix formation persists or even increases (Orban *et al.*,[20] 1944). Beust[21] believed that formation of transparent and opaque dentin was a normal growth phenomenon, but that trauma, erosion and caries could accelerate the process.

Black found that the specific gravity of teeth increased with age, but Hodge[22] believes that little change occurs between 20 and 60 years. As in other body tissues, there is gradual desiccation of the dentin. Summing up the changes which occur in dentin, Robinson *et al.*[8] conclude: "It appears that the chief aging changes in dentin are sclerosis of tubules, increase in bulk, loss of strength and possibly increase in specific gravity and mineral salts."

Pulp.—The pulp undergoes the same changes as occur in similar tissues elsewhere in the body, plus some that are peculiar to itself. The pulp, with typical pulp cells, a rich nerve and blood supply and an intact odontoblastic layer, is found only in the teeth that have been erupted for a short time, rarely in the teeth of an adult, and never in old teeth. The atrophic changes that occur in the pulp include a gradual reduction in the number of connective tissue cells which form a syncytium in the young pulp and an increase in the fibrous components of the pulp. This may result in fibrosis of the pulp and reduction in the vascular and nervous elements. In old teeth, the odontoblasts become separated by the intercellular accumulation of fluid. Kronfeld[16] describes the accumulation of fat droplets in the fibroblasts, odontoblasts and walls of the capillaries as one of the earliest changes to occur in the pulp. Calcification, as indicated by the formation of discrete nodules or diffuse deposits of calcium, is of frequent occurrence in fully formed teeth, and increases with age, occurring in unerupted teeth as well as those in function.

Metamorphosis of the dentin, formation of secondary dentin and pulp atrophy, occurring simultaneously, result in a decrease in vitality and sensitivity of the dentin and pulp with aging.

Cementum.—Abrasion of the crowns of teeth is compensated for by deposition of cementum at the apical end and bifurcation areas of the roots. This secondary cementum is normally deposited slowly and continuously throughout life, as evidenced by the presence of incremental lines (lamellae). In the recently formed peripheral layers of cementum, the cementum cells stain well and fill the lacunae; while, in the deeper, older layers, the cells stain poorly and are shrunken or have disappeared. Excessive growth of cementum is abnormal, as is formation of small isolated bodies of cementum in the periodontal membrane (cementicles). The latter is probably the result of degeneration and calcification of epithelial cells. Resorption of cementum of permanent teeth is pathologic. Gottlieb[23] says that if cementum does not continue to form, the extra-alveolar leverage is increased, as a result of

19. Bodecker, C. F.: Consideration of Some of Changes in Teeth from Youth to Old Age. D. Cosmos 67:543, June 1925.

20. Orban, Balint, et al.: Oral Histology and Embryology. St. Louis: C. V. Mosby Co., 1944.

21. Beust, T. B.: Posteruptive Changes in Maturation of Teeth. J.A.D.A. 18:2186, November 1931.

22. Hodge, H. C.: Microhardness Studies on Transparent Dentine. Brit. D. J. 63:181, August 16, 1937.

23. Gottlieb, B.: Personal communication to author.

continuous eruption, until we become physiologically edentulous.

Periodontal Membrane.—Function and age are two extremely important factors in changes in this organ. Increased function results in thickening of the periodontal membrane; reduced function, in a thinning of the membrane. The same alterations described for blood vessels, nerves, connective tissue cells, fibers, etc., elsewhere in the body also occur in these components of the periodontal membrane. The only tumor of dental origin, the ameloblastoma, is thought to develop from epithelial remnants of Hertwig's sheath in the periodontal membrane. It occurs most commonly before the age of 35.

Alveolar Bone. — The same age changes occur in alveolar bone as in other bones in the body; that is, increased brittleness, decreased vascularity, lowered metabolism and lessened ability to heal. Any abnormality or disturbance of function may result in bone changes which can be seen clinically, microscopically and roentgenographically. The dense bone immediately surrounding the teeth (lamina dura) is less readily affected than the cancellous supporting bone of the jaws, which may be influenced by nutrition and other systemic conditions, including aging. The extraction of several or all of the teeth results in extensive resorption of the alveolar bone, since the latter cannot exist without the presence of teeth. Interproximal attrition, leading to mesial drift, results in resorption of alveolar bone when the periodontal fibers are slackened on the mesial aspect and apposition of bundle bone on the distal aspect when the fibers are under increased tension. Normally, with advancing age, there is resorption of the alveolar crests. If occlusal attrition parallels the alveolar atrophy due to aging, periodontal disease and accelerated tooth loss may be avoided. Atrophy of the alveolar bone may proceed at a markedly variable rate, and the lower the rate of atrophy, the better the prognosis for retention of the teeth.

Oral Mucosa.—The mucous membrane may become atrophic in old persons, and have a smooth glazed appearance, the result of degenerative changes in the salivary glands. The formation of small retention cysts of the labial and buccal glands results from temporary cessation of function of atrophic secretory cells of those glands. This allows the duct epithelium to fuse, and subsequent stimulation of the secretory cells results in retention of the secretion and cyst formation. (Robinson *et al.*[8]) If xerostomia is extreme, grayish white patches may form on the mucous membrane, further evidence of degeneration of the oral epithelium. Ability of connective tissue to heal is greatly reduced, and loss of elasticity seems to result from actual degeneration of the elastic connective tissue and a decrease in subcutaneous fat. Loss of elasticity may also be effected by shrinkage and condensation of other intercellular substances.

Gottlieb[24] (1921) described and illustrated the organic connection of the gingiva with the tooth, calling it the epithelial attachment. In young persons, this attachment is between the gingival epithelium and the enamel cuticle. The depth of the gingival crevice varies from zero to several millimeters. With aging, the gingival epithelium proliferates apically until it is attached only to the cementum of the root. The nature of this attachment is not entirely clear. The downward proliferation of the epithelium is related to changes in the periodontal membrane and alveolar bone, and all these concomitant changes may be considered a continuation of the eruption process. Gottlieb believes that loss of the teeth with age depends on the rate of

24. Gottlieb, Bernhard: Der Epithelansatz am Zahne. Deutsche Monat. f. Zahnhlk. 39: 142, March 1921.

epithelial downward growth and the length of the root, which he calls the "reserve." It is generally recognized that the gingivae recede with age and are more prone to injury and heal less readily.

Mandible and Maxilla.—There are definite alterations in the size and shape of the face in old age, due, to some extent, to changes in the mandible and maxilla as a result of a decrease in the vertical dimension after the loss of several or all of the teeth. This is accompanied by alterations in size and shape of the maxillary antrum. There is an increase in the angle between the ramus and the body of the mandible, tending toward the condition seen in the infant. The adult type of ramus and angle can be preserved by maintaining the desirable intermaxillary dimension with correctly fitted dentures or other restorations. (Rogers and Applebaum.[25]) At the present time, we are unable to say how much of this facial change is the result of physiologic aging and how much the result of specific pathologic conditions as yet undiagnosed.

The Tongue.—The relationship of the salivary glands to xerostomia has been mentioned under the section on the oral mucosa. The decrease in volume of saliva secreted by the aged may be the result of degenerative changes in the cells lining the intermediate ducts of the salivary glands (Goodpasture[26]); cells which are considered by Stormont[27] to be the chief contributors of water to the saliva. Atrophy of the papillae of the dorsum of the tongue is associated with aging. There may be marked reduction in filiform papillae and a decrease of fungiform papillae also. This surface change, which is usually most marked at the center of the tongue, is accompanied by thinning of the mucous membrane at the borders. Occasionally, the entire dorsum of the tongue may show atrophy of the papillae. The voluntary muscle and connective tissues of the tongue, as well as the blood vessels and nerves, probably undergo degenerative changes with age as described for these tissues elsewhere in the body.

Temporomandibular Joint.—Alterations in the denture may cause disturbances of the temporomandibular joint, which may be relieved by restoring the intermaxillary dimensions to the normal. Shapiro and Truex[28] stress the fact that only where audiometric tests indicate deafness of an obstructive type will the conditions be relieved by opening the bite. A decrease in auditory acuity has been shown to occur with aging, and such change cannot be altered by dental restorations.

Caries, Periodontoclasia and Malignancy.—In addition to the conditions already described, the relation of caries, periodontoclasia and cancer to age must not be overlooked. Bodecker[29] classified dental caries as acute crown caries, chronic caries and acute root caries. The last usually affects the teeth of people past middle age, possibly as a result of food impaction around atrophic periodontal structures.

Caries is most destructive during

25. Rogers, W. M., and Applebaum, Edmund: Changes in Mandible Following Closure of Bite with Particular Reference to Edentulous Patients. J.A.D.A. 28:1573, October 1941.

26. Goodpasture, E. W.: Anatomical Study of Senescence in Dogs with Especial Reference to Relation of Cellular Changes of Age to Tumors. J. M. Res. 38:127, May 1918.

27. Stormont, D. L.: Salivary Glands, in Special Cytology, by E. V. Cowdry. New York: Paul Hoeber & Co., 1932.

28. Shapiro, H. H., and Truex, R. C.: Temporomandibular Joint and Auditory Function. J.A.D.A. 30:1147, August 1, 1943.

29. Bodecker, C. F.: Fundamentals of Dental Histology and Embryology, Including Clinical Applications. Ed. 4. New York: Columbia University, School of Dental and Oral Surgery, 1944.

youth and young adulthood, and, as its incidence decreases, periodontal disease becomes increasingly prevalent. Arrest of dental caries seems to be more common in older people. Gottlieb [24] feels that, in dental prophylaxis, we are not basically attempting to prevent increase in the depth of the periodontal pocket, but rather to prevent the loss of the tooth.

In a study of 575 oral tumors and tumor-like lesions, Darlington [30] reported almost twice as many in males as in females, and the majority of these neoplasms appeared after 40 years of age and many not until after the sixtieth year. Possible factors in the formation of buccal neoplasms include poor oral hygiene, the presence of broken-down teeth and heavy calculus deposits, ill-fitting dental prostheses and severe periodontal disease.

Discussion

Fishbein [1] writes, "Most important in prolonging life after 50 are sound rules of diet and personal hygiene and suitable attention to the detection of degenerative diseases at the earliest possible moment." The study of the various changes associated with aging (gerontology) has as yet not progressed very far. There is great need for carefully controlled research into the biology of aging. Many of the conditions now considered physiologic may be proved at some later time to result from specific diseases or pathologic states. Gerontology is important to society as a whole as well as to the individual, and further work will be necessary to solve its underlying problems.

And what of gerodontology? Are we, in dentistry, thoroughly aware of the alterations that occur in the aging masticatory apparatus? We cannot as yet state certainly whether loss of the teeth should be considered a normal involutionary change. Warthin [31] feels that loss of the permanent teeth should not commence before the age of 60. Becks and Morgan write:

> To be sure, the loss of teeth due to senescence is of a different character from that due to caries, pyorrhea, alveolar abscess, etc. However were it not for these pathologic changes in the teeth and gums that are not believed to be caused by improper diet and systemic disease, the teeth would have remained to undergo the regular involutionary changes of aging.

Many of the changes that we observe in the oral tissues depend on the presence of teeth. Caries, periodontoclasia and gingivitis do not exist in the edentulous mouth. The oral secretions are more often neutral or alkaline in the edentulous mouth than when teeth are present. However, certain pathologic conditions may attack the alveolar bone and soft tissues of the oral cavity whether teeth are present or not.

Further studies will require the coordination of all phases of basic science knowledge, together with careful observation on the part of persons well trained in all clinical aspects of dentistry. All fields of research must be utilized and coordinated to unravel the mystery of what happens to the oral tissues as man approaches his three score years and ten.

30. Darlington, C. G.: "So-Called" Tumors of Special Interest to Dentist. D. Cosmos 75:652, July 1933.

31. Warthin, A. S.: Old Age, the Major Involution. New York: Paul Hoeber & Co., 1929.

HARD TISSUE LESIONS OF THE MOUTH

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The many explanations of the cause of tooth decay collect year by year until the very multiplicity of words is overwhelming. Long ago, the great scientist Bacon spoke of "that strange distemper of learning when men study words and not matter." We must avoid such a pitfall if our profession is to continue to merit the esteem in which it has been held. The practicing dentist must insist that credit be given to his years of observation by those who are working in the field of nutrition and medical research.

The general scientist considers the teeth as a whole, responsive in varying degree to diet, environment and heredity. The dentist sees each tooth as a unit and is concerned with salvaging of the individual tooth, to the best interest of the patient. The variation in response of certain teeth or groups of teeth to disease is a commonplace observation of the dentist. The literature abounds in evidence of the reaction of the hard tooth structure to chemical agents, and the existence of one carious tooth in a mouth can be neither explained nor given proper statistical weight by those who insist on the general initiation of hard surface lesions by diet or by predilection.

The average dentist would agree with W. D. Miller's statement[1]:

> The extent to which any tooth suffers from the action of the acid (of decay) depends upon its density and structure but more particularly upon the perfection of the enamel and the protection of the neck of the tooth by healthy gums. What we might call the perfect tooth would resist indefinitely the same acid to which a tooth of opposite character would succumb in a few weeks.

1. Miller, W. D.: Independent Practitioner 4:629, 1883.

Gerald J. Cox,[2] in his "Critique of the Etiology of Dental Caries," says that this statement can be regarded as "an opinion." The confusion is one of interpretation; and as long as our profession persists in calling all lesions of the hard surface "carious," the error will persist.

The accompanying table is offered in identification of the separate groups into which lesions of the hard dental surfaces may fall. It is well to remember that each of these conditions may exist alone or all these conditions may exist at different periods in the same mouth.

Dissolution of enamel is often encountered in dental practice, and it does not seem necessary to elaborate on the effect of eating such acid-producing materials as hard candies, but the effect on the teeth of regurgitation of acid stomach contents, due to illness or anatomic peculiarities, is often overlooked. Thoma gives some excellent illustrations of the complete destruction of palatal and occlusal tooth surfaces in his description of general mouth acidity.[3] Morning sickness of pregnancy is far more likely than calcium or phosphorus withdrawal to cause dissolution or activate other lesions. Every patient suffering from this type of destruction will furnish some clue to the activating agent. In a recent case of complete and progressive loss of the palatal and lingual tooth surfaces, the patient finally mentioned that she had been taking the juice of two lemons (pH 2.2) daily in an effort to check an arthritic condition. A list of common

2. Cox, G. J.: Vitamins and Hormones. Vol. 2. New York: Academic Press, Inc., 1944, p. 272.

3. Thoma, K. H.: Oral Pathology. St. Louis: C. V. Mosby Co., 1941.

Lesions of Enamel

Condition	Under 20 Years	Over 20 Years	Area	Surface	Sensitivity	Cause	Treatment	Recurrence
Dissolution	Rare	Active	All	Soft	Slight	Chemical agents	Correction of cause	Unlikely
Attrition	Rare	Active	Occluding surfaces	Hard	Slight	Mechanical agents	Correction of cause	Unlikely
Abrasion	Rare	Active	Facial gingival	Hard	Slight	Abrasive agents	Correction of cause	Unlikely
Erosion	Rare	Active	Facial gingival	Hard	Intense	Unknown	Drug therapy or filling	Likely
Caries	Active	Less	Food retention areas	Soft	Slight	Unknown	Filling	Unlikely

canned fruits and juices[4] gives three as having a pH below 3, fifteen below 4 and eleven below 5. While extreme or continuous exposure to such acid material may be modified by the buffers of the saliva or other included materials, there can be little doubt that saturation of impacted food particles of starch or sugar would lead to decrease in the local pH. The older men of the profession well remember the practice of administering free hydrochloric acid and iron, which was followed by generalized decalcification of the teeth. The popular carbonated drinks carrying citric acid may also be assumed to add to the acidity of stagnating food particles. Fosdick[5] says:

The main chemical difference that exists between the mouths of caries-active and caries-immune people can be found in the rate of acid formation. . . . There may exist an anatomically perfect mouth which may have insufficient saliva to neutralize the acids which may be present.

This brings into evidence the fact that dysfunction of the salivary glands (as xerostomia) "leads to complete and total loss of the enamel with no respect to form or contact." (Zaus and Teuscher.[6]) Thus, chemical dissolution following contact with stomach or food acids in sufficient concentration can be considered a possible factor in decomposition of food particles and quite capable of causing destruction of all hard surfaces.

Attrition due to heavy muscular pressure or chewing of hard foods is more often found in primitive peoples free from caries. Rosebury and Karshan, commenting on "Caries in Eskimos,"[7] say, "Appreciable attrition and deposits of calculus were inversely correlated with the incidence of caries. The relationship with caries, however, appeared to depend entirely upon age." Since the

4. Canned Food Reference Manual. New York: American Can Company, 1939, p. 181 (Table III).

5. Fosdick, L. K.: Northwest. U. Bull., Spring 1943, pp. 4-5.

6. Zaus, J., and Teuscher, G. W.: J. D. Res. 19:326, June 1940.

7. Rosebury, Theodor, and Karshan, Maxwell, in Dental Science and Dental Art. Philadelphia: Lea & Febiger, 1938, p. 251.

mechanical agents necessary to cause attrition presuppose strength and age, the individual significance of these agents is easily determined. In passing, it is well to mention that the freedom from caries of primitive people may as well be due to the persistence of general stability as to any factor of the diet.

Abrasion due to improper use of the toothbrush and the use of abrasive dentifrices has received much critical attention. Granting that abrasion can initiate hard surface lesions, we should not assume that all faciocervical destruction is due to this process. Many persons that rarely use a toothbrush and many that use the toothbrush properly may suffer from similar conditions, due not alone to abrasion, but also to its close companion, erosion. Dissolution can also deepen the surface lesions and may well increase the mechanical effect of abrasion.

Erosion is the dentist's particular problem in adult mouths. It is found in clean mouths more often than in dirty ones; more often in women than in men, and more often in middle age than in youth. The sensitive, saucer-shaped areas appear gradually on the facial surfaces of the teeth at the gum margin. The dentist is impressed by the appearance of these lesions in the mouths of the more difficult patients, the tense, hypersensitive and more emotional persons presenting the utmost difficulty in treatment. There may be no question of illness, but a vague restlessness and unhappiness stemming from an emotional or a physical disorder. Typical cases are that of the woman who returns from Reno with twenty deep and painful cervical erosion cavities and that of the transatlantic pilot who develops a continuous row of erosion cavities so painful that he cannot eat hot, cold or acid food. To attribute such modification of tooth structure to diet or abrasion is manifestly unreasonable.

That dissolution through the continued application of acidic material can cause general decalcification has been noted; but, in erosion, the lesions are local, well defined and generally on both sides of the mouth. What are the local factors in this condition? The most obvious is the limitation of erosion cavities to surfaces exposed principally to the thin serous secretion of the parotid gland. Box[8] expressed the opinion that the surfaces of the teeth are seldom subject to a salivary pH environment representative of a thoroughly mixed saliva and that erosion is a preeminent lesion of the oral vestibule. This conception is of tremendous importance if the steps leading to erosion are to be visualized. The alternative conception that mixed saliva is indicative of oral health is erroneous and delusive.

Rabkin,[9] in describing the considerable disagreement existing among physiologists regarding the function of the salivary glands, states that if the human salivary glands secrete spontaneously and various stimuli serve merely to enhance their secretion, in this respect man stands alone among the higher vertebrates. The Russian scientist Pavlov, in his physiologic studies in experimental neuroses on dogs, based his findings, to a great extent, on the rate of salivary flow following excitatory and inhibitory conditions. Barrett[10] has demonstrated in human beings that, in emotional crises, the pH of the saliva may fluctuate rapidly, finally dropping to a constant acid level. Bunting[11] writes:

There is ample evidence that systemic conditions play an important role in the

8. Box, H. K.: Bull. 24. Canad. D. Res. Foundation, 1938, p. 15.

9. Rabkin, B. E., in Dental Science and Dental Art. Philadelphia: Lea & Febiger, 1938, p. 222.

10. Barrett, L. G.: Semantogenesis and Dental Caries. Unpublished data.

11. Bunting, R. W., and Hill, T. J.: Oral Pathology. Philadelphia: Lea & Febiger, 1940, p. 154.

chemical erosion of the teeth. It has been observed by one of us (R.W.B.) that it occurs more commonly in individuals who are alternately under high nervous strain and who in the pressure of their occupation or their pleasures expend considerable nervous energy.

Wiggers[12] describes a purely nervous form of xerostomia during the menopause.

The conception of disease as a fixed state is gradually being abandoned by medicine. Local disease following emotional strain has been demonstrated in stomach ulcers and many gastro-intestinal disorders. The addition of cervicofacial erosion of the teeth to this list is reasonable. The dentist cannot be expected to treat neuroses, and the patient is interested primarily in relief from local discomfort. Thus, as later described, the treatment of erosion must be entirely local and directed to the relief of symptoms.

Caries may, in some way, be connected with nutritional disorder. The tremendous amount of information collected to date challenges our powers of analysis. Virtually all of the identified growth factors or "vitamins" have been connected with development and structural perfection of the teeth or their supporting structures. The acid-base composition of the whole diet may accelerate or hinder caries. Yet little information having practical application has been furnished by the dentist. Boyd[13] maintained that an adequate diet would limit the disease, while Ziskin *et al.*[14] deny that this finding is correct. The statistical studies of East[15] seem to show that in the northern and eastern sections of our country dental ills are more common than in the southern. McBeath and Zucker[16] have advocated the reinforcement of the diet with vitamin D. Normal expectation would be that the "perfect tooth," as previously described by Miller, or the "anatomically perfect mouth," as described by Fosdick, would be one in which the calcium-phosphorus: vitamin D ratio had been maintained at all times. The advocacy by the dentist of an adequate diet must not cease merely because he is not sure of the particular condition responsible for dental health.

When perfection of study leads to perfect prophylaxis or prevention of dental disorders, there will be no need for our services. Until then, we must continue our local treatment. The elimination of abrasion and attrition can be carried out by the removal of the "cause" when possible. Chemical or acidic agents contributing to dissolution and aggravating the other disorders must be removed; but, for the repair of local lesions, we must depend on final filling together with such general agents as we now have at our disposal. These take the fashion of agents capable of recalcifying or hardening local areas. In 1922-1925, Viggo Andresen[17] published an exhaustive study on the "Physiological and Artificial Remineralization of the Enamel." Whether remineralization of the enamel is possible is a disputed point, but the fact remains that, through the use of a "remineralizing powder," it seemed to be possible to make tooth surface lesions more resistant and less sensitive. Silver nitrate and zinc chloride have been applied topically with

12. Wiggers, C. J.: Physiology in Health and Disease. Philadelphia: Lea & Febiger, 1937, p. 766.

13. Boyd, J. D.: Role of Diet in Control of Dentinal Caries. J.A.D.A. 27:750, May 1940.

14. Ziskin, D. E.; Siegel, E. H., and Loughlin, W. C.: J. D. Res. 23:399, October 1944.

15. East, B. R.: Am. J. Pub. Health. 32: 1242, November 1942.

16. McBeath, E. C., and Zucker, T. F.: J. Nutrition 15:547, June 1938.

17. Andresen, Viggo: Physiological and Artificial Remineralization of Enamel. Oslo. 1922-1925.

the same view. Lukowsky[18] described a method of applying sodium fluoride directly to the eroded or carious surfaces (sodium fluoride, 1 part; white clay, 1 part; glycerin, 1.2 part). Souder and Schoonover[19] reported remineralization *in vitro* of tooth structure using sodium fluoride and calcium sulfate. Sodium fluoride tends to hydrolyze in the presence of moisture and must be kept in a tightly sealed container. Elaboration of this treatment seems to offer the most effective means of treating caries or erosion. In the use of fluorine, care must be taken not to approximate a toxic dose, and every precaution must be used in applying so toxic a substance. The inclusion of sodium fluoride in mouth washes, dentifrices or drinking water is much more debatable. As Bibby[20] has pointed out: "The importance of our investigations on the control of dental decay by the use of fluorides may not be so much that it demonstrates a method of reducing caries as that it points to a new principle in caries control."

Conclusion

Distinction must be finely drawn among dissolution, attrition, abrasion, erosion and caries.

Dissolution, attrition and abrasion, which follow definite patterns, may be treated by removal of the aggravating agents.

Erosion is limited to surfaces exposed to the parotid saliva, and, while the initiation of the lesion is as yet unexplained, there is reason to believe that emotional and physical stress contribute to the disorder.

Caries is limited to food stagnation surfaces and the process of development is as yet unexplained.

Caries and erosion may be treated through artificial remineralization as with Andresen's powder or by local application of sodium fluoride, in addition to filling where necessary.

Elimination of foods of low pH must be insisted on in patients showing high caries susceptibility, until the mouth is rendered mechanically free of surfaces where food is retained.

Time is a factor in all lesions of the hard dental tissues. The conception of these conditions as definite "disease" should be abandoned and continuous dental attention directed toward the removal of "cause," limitation of mouth acidity of local origin and restoration to normal physical form by dental treatment.

350 Madison Avenue.

18. Lukowsky, E. H.: J. D. Res. 20:649, December 1941.

19. Souder, Wilmer, and Schoonover, I. C.: Experimental Remineralization of Dentin. J.A.D.A. 31:1570, December 1, 1944.

20. Bibby, B. G.: Fluorine and Dental Caries. New York J. Dent. 15:103, March 1945.

ANATOMY OF THE FACE AND MOUTH FROM THE STANDPOINT OF THE DENTURE PROSTHETIST*

Elbert C. Pendleton, D.D.S., M.D.S., Chicago, Ill.

A study of the gross anatomy of the denture-supporting tissues was undertaken in the winter of 1941 and 1942. Up to that time, the primary objective had centered in a study of minute anatomy. It had been suggested that the student of denture prosthesis found it difficult to orient histologic specimens with the clinical picture. It was the hope that dissections of the human jaws might make the minute structures more comprehensible.

It is, therefore, the purpose of this study to present the anatomy of the face and mouth from the operator's approach to the patient so that the structures associated with the denture problem may be projected in their true perspective. MacMillan pointed out that "textbook descriptions of the muscles of the normal mandible that contains teeth do not apply to the muscles of the edentulous mandible."[1] This observation also applies to the muscles of the maxilla and the face and the muscles of mastication, which, owing to resorption and functional changes, show many variations in the edentulous state not found in jaws with natural teeth.

It has long been known that the human body is subject to many variations, notwithstanding a marked consistency in the general pattern of its tissues. Therefore, in applying these studies in clinical practice, the student may well be surprised when the anatomic structures present no unusual developments. Variations in form, relation and character of the tissues of the edentulous jaws are many and often extreme. Research investigations, both laboratory and clinical, suggest that the primary influence in tissue change, as it affects the prosthetic problem, is functional.[2] What may be referred to as normal variations are no doubt affected by racial, hereditary and environmental conditions. "Even among individuals of the same race, sex and age, variations occur in all their physical traits, including innumerable details of both the gross and microscopic structure."[3]

Early in the history of dentistry, Chapin A. Harris wrote: "The utility of artificial teeth depends upon their being properly constructed and correctly applied. . . . A knowledge of the anatomy and physiology of the mouth, of its various pathological conditions and therapeutical indications is as essential to the mechanical as it is to the operative dentist. . . . There are difficulties connected with the insertion of artificial teeth which none but an experienced dentist has any idea of."[4] This statement brings to mind a saying of J. B. Davis, quoted from a tablet in the Engineering Arch at the University of Michigan, "Young man,

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1. MacMillan, H. W.: Anatomy of Throat, Mylohyoid Region and Mandible in Relation to Retention of Mandibular Artificial Dentures. J.A.D.A. 23:1435, August 1936.

2. Pendleton, E. C.: Reaction of Human Jaws to Prosthetic Dentures. J.A.D.A. 27: 667, May 1942.

3. Jackson, C. M., and Blount, R. F.: Digestive System. Morris' Human Anatomy (Schaeffer). Ed. 10. Philadelphia: Blakiston Company, 1942, p. 7.

4. Harris, C. A.: Principles and Practice of Dental Surgery. Ed. 7. Philadelphia: Lindsay and Blakiston Company, 1858, p. 659.

when theory and practice differ, use your horse sense."[5]

Basic training in the sciences is fundamental to good judgment. Thomas Huxley has been quoted as defining science as "trained and organized common sense." The construction of artificial dentures requires trained and organized judgment in the transfer of theory to practice, where the operator's resourcefulness is often the decisive factor in success.[6]

George Henry Wilson said, "No one factor has so much to do with retention of artificial dentures as the soft tissues. These may be divided into three classes, muscles and their attachments, submucous tissue and the mucous membrane."[7]

The stabilizing effect of the muscles of the lips and cheeks, as well as the muscles of mastication, on the denture is emphasized by Fish.[8] Boos said, "The muscles are the basis of facial expression as well as a part of the facial contour."[9]

Authors, past and present, are of one accord: the mechanical solution of the problem of denture prosthesis is dependent largely on an understanding of its biologic relationship.[10]

The Face

The surface of the face is covered by skin, which consists of two main layers, the surface epithelium and a dense connective tissue (corium). The facial muscles of expression have their chief attachment in the dense connective tissue.[11] Supporting this dense connective tissue is a loose connective tissue that may be transformed into a subcutaneous fatty tissue, richly supplied with blood vessels and nerves.[12] Fatty tissue is not uncommon about the face and its presence adds materially to the difficulties encountered in preparing dissections of the cadaver. The terminal fibers of the muscles of the lips and chin commonly cannot be separated from the extremely dense fibrous connective tissue of the skin in the male subject, because of its character. The task is insurmountable when an abundance of fatty tissue is encountered.

Dissections of the facial muscles were prepared so that, in right profile, left profile or frontal view, each muscle might be illustrated. For example: The superior quadratus muscle was incised on the right side of the face so that the caninus muscle could be seen (Fig. 1*A*). On the left side of the face, the caninus, zygomaticus, risorius, triangularis and platysma muscles were either cut or removed in order to expose the inferior quadratus, buccinator and masseter muscles and the parotid gland with its duct (Fig. 1*B*). The mentalis and the inferior incisive muscles, illustrated in Figure 1*C*, are shown in the frontal view.

The chief interest of the denture prosthetist centers in and around the mouth. Its muscle action is complex and, if properly employed, will aid in the retention and stability of artificial dentures.[13]

5. Davis, J. B.: Post War Program, General Bull. 42, University of Michigan, Vol. 45, No. 132, May 2, 1944.

6. Lowery, P. C.: Esthetic Consideration in Construction of Artificial Dentures. American Textbook of Prosthetic Dentistry (Anthony). Ed. 7. Philadelphia: Lea and Febiger, 1942, p. 386.

7. Wilson, G. H.: Principles of Retention of Artificial Dentures. D. Cosmos 51:934, August 1909.

8. Fish, E. W.: Principles of Full Denture Prosthesis. Ed. 2. London: John Bale Sons and Danielsson, Ltd., 1933, p. 1.

9. Boos, R. H.: Intermaxillary Relation Established by Biting Power. J.A.D.A. 27: 1192, August 1940.

10. Sears, V. H.: Basic Principles in Dentistry. New York: Pitman Publishing Corporation, 1942, p. 22.

11. Cummins, Harold: Skin and Mammary Glands. Morris' Human Anatomy (Schaeffer). Ed. 10. Philadelphia: Blakiston Company, 1942, pp. 60-61.

12. Maximow, A. A., and Bloom, William: Textbook of Histology. Ed. 3. Philadelphia: W. B. Sanders Company, 1938, p. 323.

13. Fish, op. cit., pp. 5, 8, 100.

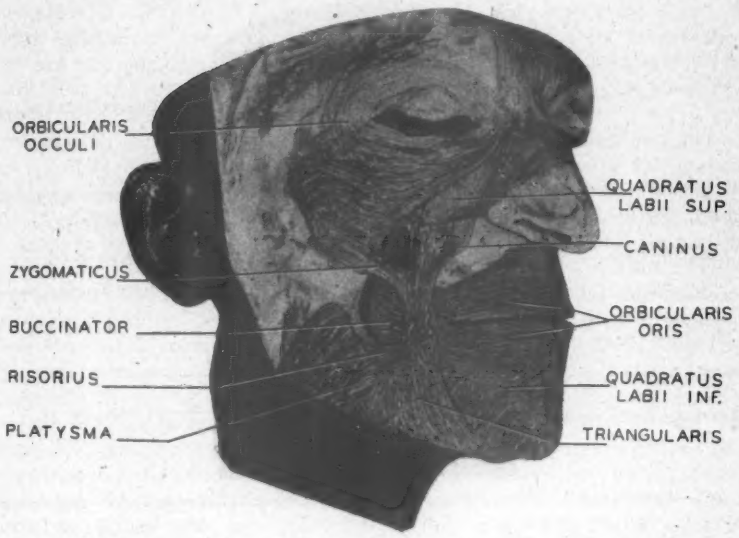

Fig. 1*A*.—Superior quadratus cut to expose caninus muscle.

The muscles of the mouth, most of which have an osseous origin and cutaneous insertion, belong to the facial system. There are exceptions. Both origin and insertion may be cutaneous (the orbicularis oris) or their attachment may be osseous and aponeurotic (the buccinator), and the deep musculature about the mouth has its attachment in the mucosa. The interdigitating fibers

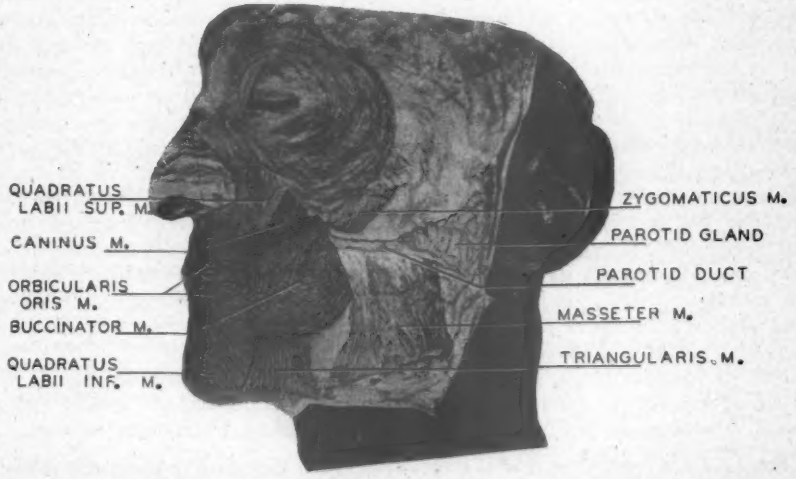

Fig. 1*B*.—Zygomaticus, caninus and triangularis muscles cut, platysma, risorius and superficial fascia removed, to expose masseter and buccinator muscles and parotid gland and duct.

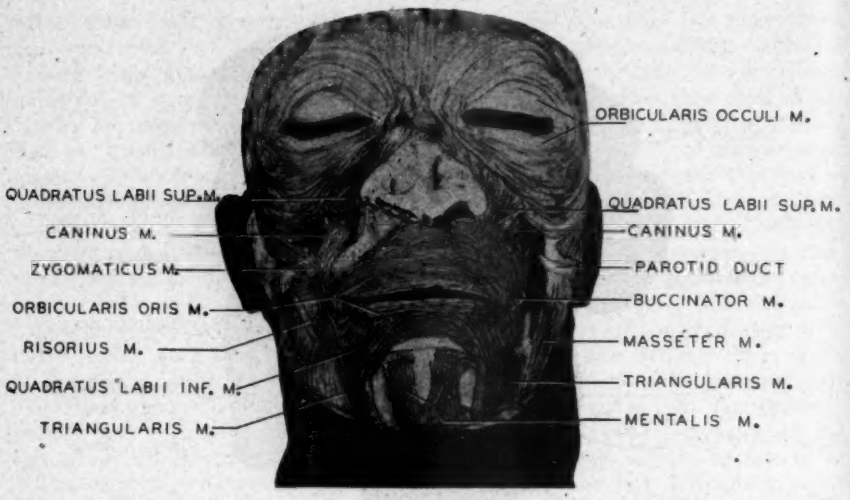

Fig. 1C.—Portions of inferior quadratus and triangularis muscles removed to expose mentalis muscle.

of the facial muscles have a parallel or slightly converging course. Their complex development varies. From each corner of the mouth, several muscles radiate upward, downward and laterally over the cheek and side of the oral cavity. From each of these, the caninus, zygomaticus, platysma, triangularis, risorius and buccinator, fiber-bundles are continued into the orbicularis oris.[14]

14. Grant, J. C. B.: Musculature. Morris' Human Anatomy (Schaeffer). Ed. 10. Philadelphia: Blakiston Company, 1942, pp. 393-395.

Fig. 2.—Facial muscles cut close to bony attachment to show areas of labiobuccal surface of upper and lower jaws directly affected by musculature in taking impressions and adapting denture borders.

Movements of the lips and cheeks are controlled by individual and united action of one or more of these muscles. It seems that the orbicularis oris and buccinator assisted by the caninus and triangularis may be of the greatest influence in the denture problem. The incisive muscle may have a nuisance value. It, like the mentalis, which is often strongly developed, may combine its action with that of the orbicularis in the protrusion of the lips.

The osseous origin of the facial muscles, affecting the labiobuccal borders of the denture, is restricted. Their clinical significance is limited by the relation of the muscle bundles to the periphery of the denture. Though the potential force of their action may be extreme, this force is minimized by the presence of the greater area padded with fat-contained connective tissues free from muscle attachments. This muscle-free area acts as a buffer, aiding in neutralizing the forces affecting the stability of the denture. Figure 2 shows these surfaces of the denuded bone and the muscles cut close to their point of origin. It is revealing that the bare dissection should expose the buccinator as the one muscle of appreciable concern to the prosthetist. The incisive, depressor nasi and mentalis may, in cases of extreme resorption, have fibers close to the denture border. It appears that the end fibers of the facial muscles having attachments to the mucosa at the labiobuccal vestibule are the main source of annoyance in stabilizing the denture base. These are the muscle attachments that control the movements and affect the adaptive contacts of the mucosa with the periphery of the denture in the "zone of mucosal flexion."[15]

In the development of retention and stability of the prosthetic denture, contact must be maintained by the appliance with the supporting tissues. The border tissues must not disturb the balance of the denture in its relation to the supporting tissues or else stability, and then retention, is lost.

A visual examination may not reveal the degree of action or the amount of compressibility of the tissues of the vestibule. The roll that identifies both the zone of flexion and the border line of the periphery of the denture may be interpreted best in the impression. It is essential to the retention of the artificial denture and to successful treatment that the zone of mucosal flexion be determined. According to Sears,[16] one gains a more accurate idea of the border tissues by the interpretation of an impression made with free-flowing materials inside well-fitting trays than it is possible to gain by direct examination of the oral tissues.

The Edentulous Jaws

The hard and the soft palate form the roof of the mouth. The hard palate is bounded anteriorly and laterally by the residual alveolar ridges and may be divided into two parts: anterior and posterior. The anterolateral walls of the oral cavity are formed by the lips and cheeks. Between the residual ridge and the lips and cheeks is the vestibule.

The anterior portion of the hard palate is covered by a thick, dense fibrous mucosa. Its pale surface is marked by a median line or ridge (raphe), from which irregular elevations (rugae) extend anterolaterally. The soft palate is attached to the posterior border of the hard palate. Its "skeleton" is the palatine aponeurosis, which serves as an attachment for the palatine muscles and supports the palatine glands and adipose tissue that form the bulk of its structure. The surface of the soft palate is smooth, presenting suggestions of more highly vascular (pink) tissues.

15. Wright, W. H.: Method of Securing Correct Impressions. J.A.D.A. 27:1584, October 1940.

16. Op. cit., p. 20.

Dissection of the Palate

A midsagittal incision was made through the mucosa posteriorly from about the plane of the cuspid eminence, including the soft palate. Another oblique incision extended to the first molar region of the maxillary residual ridge. The mucosa, fatty tissue and palatine glands were removed to expose the palatine process of the maxillary and horizontal part of the palate bones together with the greater and lesser palatine foramina and posterior nasal spine. The structures exposed in the soft palate include the palatopharyngeus muscle, the palatine aponeurosis and a portion of the tendon of the internal pterygoid muscle.[17] (Fig. 3.)

In the succeeding dissection, the right side of the palate was treated similarly to the left and in addition the levator palati muscle was exposed. Further treatment was given the left side by reducing the posterior portion of the maxillary tuberosity and by the removal of the pterygomandibular raphe together with the buccinator and superior constrictor muscles. This operation exposed the internal pterygoid muscle and the tendinous attachments of the temporal muscle. (Fig. 4.)

The tissues of the mouth, both hard and soft, are presented for direct observation from the approach that the operator might make in the examination of an edentulous patient.

From the viewpoint of the denture prosthetist, the palate assumes huge proportions. Retention in denture construction at times is as elusive as stability is baffling. Who among us can supply the answer? Is failure concealed in some detail of technical precision, or is it not possible that there is a lack of appreciation of the anatomic conditions?

The character and distribution of the palatine tissues have been described in

17. Sears, op. cit., pp. 15-16.

Fig. 3.—Posterior border of hard palate, greater and lesser palatine foramina, maxillary tuberosity, pterygoid hamulus, tendinous attachment of internal pterygoid muscle and palatine aponeurosis exposed by removal of palatine mucosa and fatty and glandular tissues at postdam area on left side of palatine vault. Insert: Section at pterygoid hamulus of unretouched photograph from which illustration was made.

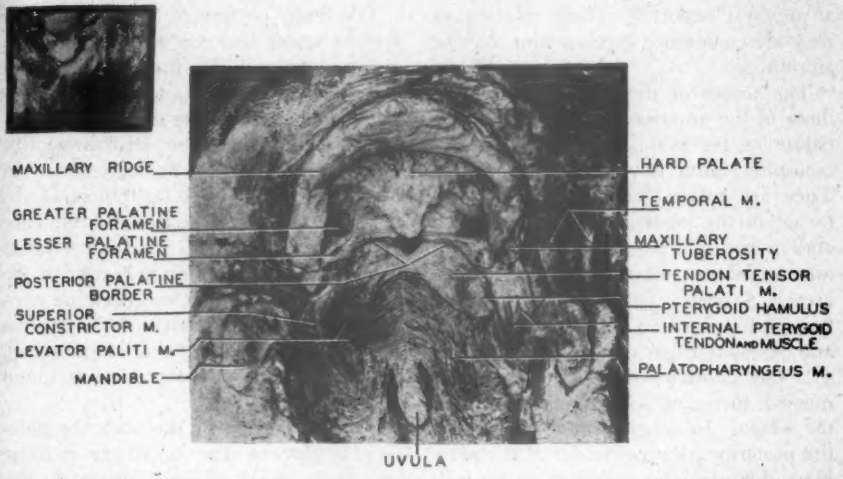

Fig. 4.—Complete exposure of postdam area showing remote relation of levator palati muscle to posterior border of hard palate, intimate relation of attachment of internal pterygoid muscle to maxillary tuberosity and extent of palatine aponeurosis. Insert: Section at pterygoid hamulus of right side of velum palati. The dark area at the lower right border shows slit where the levator palati emerges to interdigitate with the palatopharyngeus muscle.

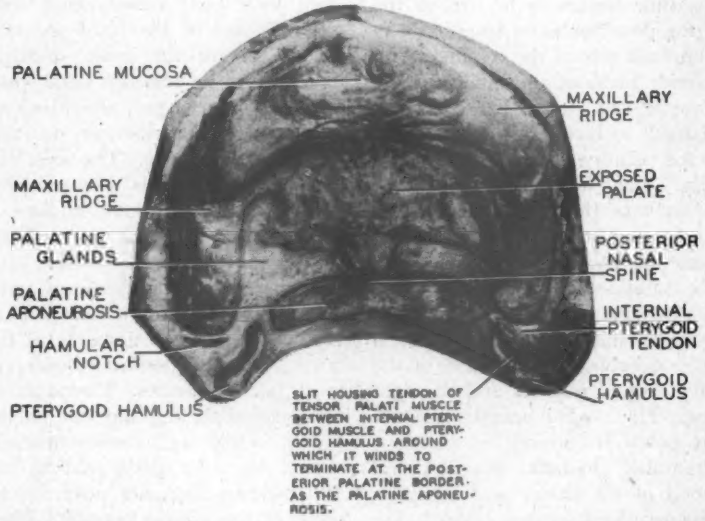
PALATINE MUCOSA
MAXILLARY RIDGE
MAXILLARY RIDGE
PALATINE GLANDS
PALATINE APONEUROSIS
HAMULAR NOTCH
PTERYGOID HAMULUS
EXPOSED PALATE
POSTERIOR NASAL SPINE
INTERNAL PTERYGOID TENDON
PTERYGOID HAMULUS
SLIT HOUSING TENDON OF TENSOR PALATI MUSCLE BETWEEN INTERNAL PTERYGOID MUSCLE AND PTERYGOID HAMULUS AROUND WHICH IT WINDS TO TERMINATE AT THE POSTERIOR PALATINE BORDER AS THE PALATINE APONEUROSIS.

Fig. 5.—Cast from impression of dissection specimen in Figure 4; showing area and depth of fatty and glandular submucosa of posterior portion of vault and postdam area, relation of pterygomaxillary (hamular) notch to maxillary tuberosity and pterygoid hamulus (hamular process) on right side of cast. The soft tissues forming the floor of the hamular notch are shown on the left side of the cast.

a previous report.[18] Their relation as they affect denture construction may be amplified.

The tissues of the residual ridge and those in the anterior portion of the hard palate as far posteriorly as the second bicuspid region have limited elasticity. They are rather firm and dense. The tissues in the posterior part of the hard and in the soft palate are compressible and extremely elastic, except in local areas. A deep submucosa may be anticipated in the area between the midline and residual ridge of the maxilla (Fig. 5). The depth or thickness of the submucosa increases with the approach of the uvula. In other words, the farther the posterior palatine border of the maxillary denture is extended upon the soft palate, the more difficult is the problem of maintaining its adaptation, owing to the progressive increase in the depth of the submucosa and the accelerated potential action of the soft palate.

Direct muscle interference at the posterior palatine border is limited to the area of the posterior nasal spine or 3 to 5 mm. on each side of the midline. The most inferior fibers of the internal pterygoid muscle are attached at the posterior lateral surface of the horizontal part of the palatine bone and maxillary tuberosity. They form the floor of the pterygomaxillary (hamular) notch. This notch is located between the tuberosity of the maxilla and the pyramidal process of the palatine bone. Its surface covering is a delicate mucosa. A submucosa that is quite shallow provides only a thin cushion between the tendon of the internal pterygoid muscles and the denture base. The medial boundary of the hamular notch is formed by the pterygoid hamulus (hamular process) and the tendon of the tensor palati muscle. This region offers another, though less potent, obstacle to denture stability.

18. Pendleton, E. C.: Minute Anatomy of Soft Palate from Viewpoint of Denture Prosthetist. Bur 44:13, April 1944.

The fleshy portion of the tensor palati muscle passes between the medial pterygoid plate and the internal pterygoid muscle approaching its tendon at nearly right angles as it passes around the pterygoid hamulus. These structures, like those of the levator palati, appear, in most instances, to be so far removed from the denture border that their influence seems negligible. Between the pterygoid hamulus and the posterior border of the hard palate to which the palatine aponeurosis (terminal fibers of the tendon of the tensor palati muscle) is attached, a variable area of adaptation is found (Fig. 4).

The adaptation of the posterior palatine border of the maxillary denture (postdam area) differs in no fundamental particular from other peripheral contacts. The tissues of the soft palate do vary greatly from other border tissues in their essential characteristics. The muscles affecting the movements of the soft palate, in the main, are far removed from their bony attachments, the posterior border of the hard palate. The exceptions are the uvula muscle with its origin in the posterior nasal spine and the palatopharyngeus, which is attached to the palatine aponeurosis on each side of the uvula muscle. The area of these attachments extends approximately 4 mm. on each side of the midline.

The mucosa of the postdam area is usually thin and quite elastic. Its submucosa has an abundance of fatty and glandular tissues forming a thick cushion that deepens proportionately as the distance from the posterior border of the hard palate increases. The palatine aponeurosis supporting the soft palate is a tough, flexible collagenous structure designed to resist great pulling forces.[19] Its thickness increases near the attachment of the muscle bundles. The muscle-free part of the aponeurosis, between the bone and muscle bundles, varies from

19. Maximow and Bloom, op. cit., p. 62.

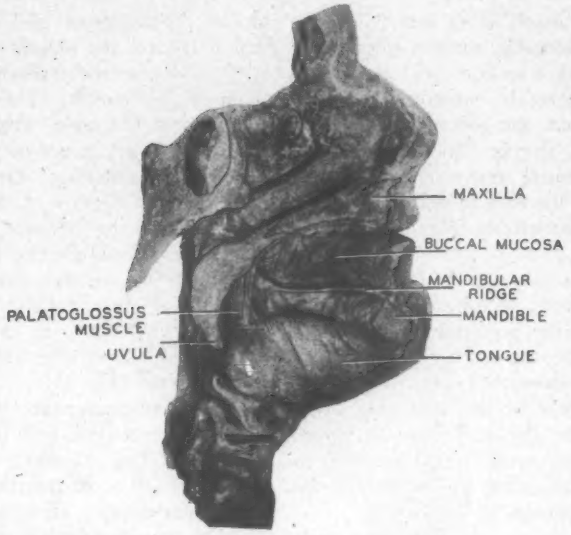

Fig. 6.—Head of cadaver sectioned in midsagittal plane; showing maxilla, buccal mucosa of oral cavity, lingual surface of mandibular residual ridge and tongue related to palatoglossus muscle.

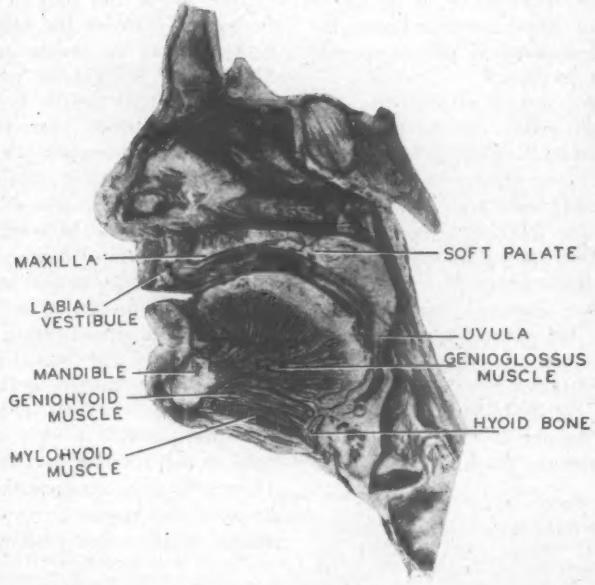

Fig. 7.—Midsagittal view of tongue related to palatine vault, lips and vestibule.

1 mm. to as much as 12 mm.[20] Therefore, a considerable surface unopposed by direct muscle action is available for denture support. In extending the denture base upon the soft palate, thicker aponeurosis, deeper submucosa and greater potential movements are encountered as the area of denture contact is increased posteriorly from the palatine border.

These facts may well be considered in the development of the adaptation and extension of the posterior palatine border of the denture. The establishment of precise extension in the impression tray, regardless of the type employed, appears to be the procedure to insure successful treatment. It is hazardous to depend on adjusting the border of the completed denture.

The Tongue

The tongue is one of the most potent factors in the stability and retention of prosthetic dentures. It may, by its action, enhance stability; or, if its movements are in the least restrained, the tongue may displace, if not completely dislodge, the dentures.[21]

The inferior surface of the body (the large anterior part) of the tongue is chiefly related to the muscles of the floor of the mouth from the hyoid bone to the mandible. Anteriorly and laterally, it is free. The free inferior surface is covered by a thin, smooth mucosa. In the midline of this surface is a prominent fold (frenulum linguae) connecting the tongue with the mandible and floor of the mouth.

Muscles of the Tongue.—The tongue is composed chiefly of muscles. The extrinsic muscles are of chief importance to the prosthetist: the hyoglossus, styloglossus, genioglossus and palatoglossus, which control the action of the tongue and influence the movements of the floor of the mouth. The genioglossus, the principal muscle, arising from the mental (genial) spines of the mandible, has fibers radiating throughout the length of the dorsum of the tongue. It is important to denture stability because of its attachments, which are invariably tendinous and, as such, are often troublesome in adapting the denture borders, particularly in extreme resorption, which accentuates the form of the mental spines (Fig. 7).

The palatoglossus muscle arises at the side of the tongue and passes upward in front of the palatine tonsil. It forms a thin sheath at its insertion in the palatine aponeurosis. Its constrictor action may be considered of slight clinical importance in prosthesis because of its remoteness from the field of operation.[22] (Fig. 6.)

The Sublingual Region.—The sublingual region is that part of the floor of the mouth between the inferior surface of the body of the tongue and the mandible. It is bridged by mucous membrane supported by the mylohyoid and geniohyoid muscles. On each side of the "frenulum linguae" are openings of the submaxillary and sublingual ducts.

The Sublingual Structures.—The structures in the space between the tongue and mandible were exposed by the removal of the mucosa and incision of the genioglossus muscle. The tongue was displaced, and the sublingual gland, submaxillary duct and lingual nerve, which are virtually supported by the mylohyoid muscle, were revealed (Fig. 8). This operation presents a view of the structures in the anterior part of the mouth. These structures are critical to the adaptation of the lingual flange of the mandibular denture, but perhaps not of the

20. Pendleton, op. cit., p. 19.

21. Gysi, Alfred: Importance of Providing Tongue Space. Special Teeth for Cross Bite Cases. D. Digest 33:39, February; 859, December 1927; 34:18, January; 182, March 1928.

22. Jackson and Blount, op. cit., pp. 1252-1257.

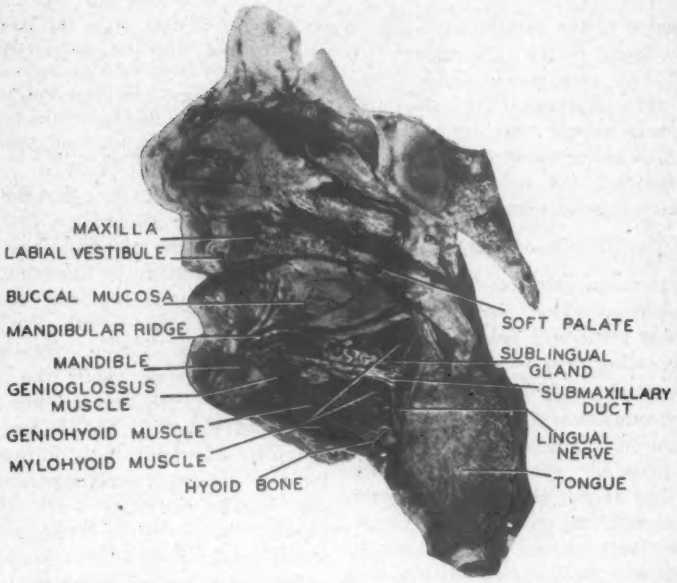

Fig. 8.—Midsagittal view of structures at lingual surface of mandible supported by mylohyoid muscle. The genioglossus muscle was cut and the tongue was drawn back to expose the structures related to the lingual flange of the mandibular denture.

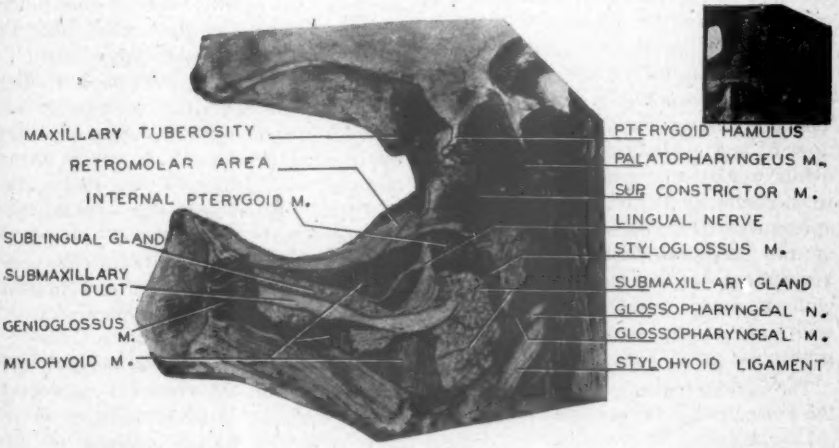

Fig. 9.—Lingual aspect of mandibular residual ridge, retromolar and retromylohyoid space, lingual surface of vault and pterygomandibular raphe. Incision at the posterior part of the retromolar papilla to the attachment of the pterygomandibular raphe indicates the depth of retromolar postdam tissues. Insert: Unretouched photograph showing portion of lingual nerve extending toward incision at the retromolar papilla, pterygomandibular raphe serving as attachment of superior constrictor muscle, and associated structures beneath floor of mouth.

same interest to the prosthetist as those structures found in the "retromylohyoid space."[23] This area was exposed by removing the pharyngeal mucosa, the palatoglossus muscle and the palatine tonsil, which rested on the superior constrictor between the palatoglossus and palatopharyngeus muscles.

The Retromolar Area

The retromolar area together with the "retromylohyoid space" constitutes one of the most vulnerable fields in denture prosthesis owing to the varied character and the function of its tissues.

The retromolar area is located at the posterior limits of the ridge of the body of the mandible where it joins the ramus. The area is almond shaped and is marked near its center by an elevation, the retromolar papilla, which should always be included within the area of denture support. The submucosa of the ridge, anteriorly from this elevation, is composed of fatty and glandular tissues and affords an area for the postdam process. Posteriorly and medially from the retromolar papilla, the submucosa is of loose connective tissue, in which fibers from the pterygomandibular raphe are encountered.[24] The pterygomandibular raphe continues upward and medially from these attachments to the pterygoid hamulus, where it becomes part of the pharyngeal aponeurosis (Fig. 9). The two tendons of the temporal muscle extend their attachment to the ramus at its union with the body of the mandible, one on each side at the posterior part of the retromolar area (Fig. 10).

The superior constrictor muscle arises from the lower third of the pterygoid process and its hamular process, from the pterygomandibular raphe, from the posterior fifth of the mandible and from the side and root of the tongue. Its fibers pass backward to be inserted into the median raphe; the lower fibers arch downward and are overlapped by the middle constrictor.[25] (Fig. 11.)

It is important to note that the portion of the superior constrictor that is said to arise from the mandible has only an indirect attachment to this bone. These fibers arise from a membrane that forms the floor of the mouth in its posterior and lateral area. The pterygomandibular raphe, with the superior constrictor muscle, covers the internal pterygoid muscle. The lower part of the internal pterygoid is intimately related to, though sharply separated from, the posterior extremity of the mylohyoid muscle. Extended insertion of the internal pterygoid muscle is found at the mylohyoid ridge. In a previous report, it was said that "the muscles of the mandible intimately related to the prosthesis and having either or both periosteal or aponeurotic attachments to the bone are . . . (5) the superior constrictor of the pharynx, in the molar, and, in rare instances, postmolar lingual area."[26] Since the completion of these studies from the dissection of a number of cadaver specimens, it is apparent that the quoted interpretation was in error and the muscle fibers "in the molar and postmolar lingual area" are in reality attachments of the internal pterygoid muscle. Their presence is of no less concern in the adaptation of the lingual flange of the mandibular denture (Fig. 9).

The lingual nerve passes between the lower part of the internal pterygoid muscle and the mylohyoid ridge, where it is near the former position of the

23. Edwards, L. F., and Boucher, C. O.: Anatomy of Mouth in Relation to Complete Dentures. J.A.D.A. 29:331, March 1942.

24. Pendleton, E. C.: Influence of Biologic Factors in Retention of Artificial Dentures. J.A.D.A. 23:1233, July 1936.

25. Jackson and Blount, op. cit., pp. 1282-1283.

26. Pendleton, E. C.: Minute Anatomy of Lower Jaw in Relation to the Denture Problem. J.A.D.A. 29:721, May 1942.

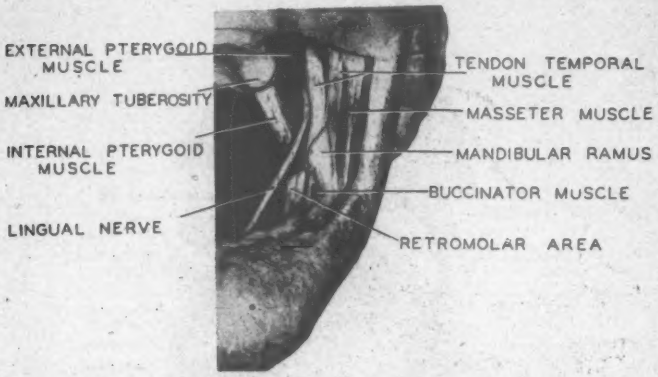

Fig. 10.—Frontal view of tuberosity of maxilla related to external pterygoid and internal pterygoid muscles and tendons of temporal muscle to retromolar area of mandible.

mandibular third molar tooth.[27] The sublingual division of the lingual nerve crosses the mylohyoid muscle, extending obliquely forward and downward to supply the sublingual gland and the mucous membrane. The sublingual nerve is pertinent to the prosthetic problem as it may be affected by the posterior and downward extension of the lingual flange of the mandibular denture (Fig. 9).

The mylohyoid muscle, stripped of its covering of mucosa, glands, ducts, nerves and fascia (the structures supporting the lingual border of the mandibular denture), arises from the mylohyoid ridge of the mandible. It supports the floor of the mouth and extends obliquely downward to form a curtain, draped between the inner surface of the mandible and the hyoid bone.[28] (Fig. 12.)

Border tissues are stimulated to activity by each movement of the lips, cheeks, mandible, soft palate, pharynx, and tongue in respiration, speech, mastication and deglutition. It is noteworthy that all changes of expression under emotional stimuli are caused by contractions of muscles of the face. Therefore, emotional stability is likewise a factor in prosthetic service and the patient's control of the musculature may contribute greatly to the success of the denture as the adaptive contacts of the prosthetic denture are affected by the border tissues that contribute to its support.

27. Larsell, Olof.: Nervous System. Morris' Human Anatomy (Schaeffer). Ed. 10. Philadelphia: Blakiston Company, 1942, p. 1059.

28. Grant, op. cit., p. 409.

Conclusions

The treatment of the edentulous condition is a problem of the control and distribution of stresses borne by the denture-supporting tissues. Two conditions, one mechanical and the other biologic, affect the stability and retention of prosthetic dentures. These are related so intimately that neither can be considered separately. The soft tissues supporting the denture are variable to the degree that failure to interpret their characteristics and tolerance may impair the results of treatment. The tissues of the face and mouth are distributed with great regularity. A lack or an abundance of fatty tissue is of considerable importance in denture service.

Glandular and fatty tissues are usually closely associated and conveniently lo-

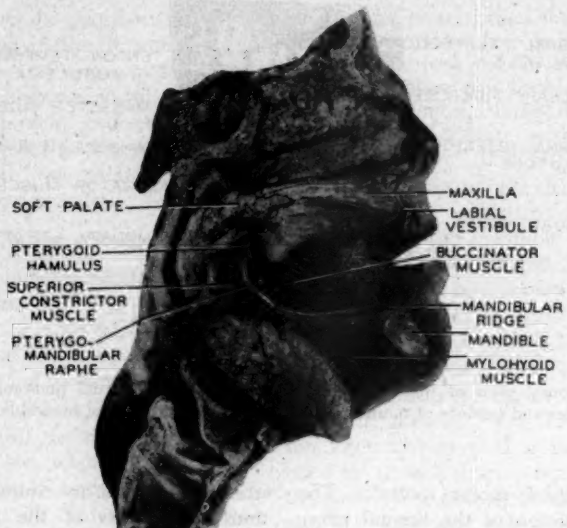

Fig. 11.—Frontolateral view of inner surface of cheek and lingual aspect of mandibular residual ridge stripped of mucosa to expose mylohyoid muscle, and buccinator and superior constrictor muscles related to pterygomandibular raphe and pterygoid hamulus.

Fig. 12.—Mylohyoid muscle stripped of covering of mucosa, muscles, glands, ducts and nerves, from mylohyoid ridge of mandible.

cated at or near the border of the denture-bearing area. Denture stability depends largely on the dissipation of stress exerted by the musculature. Fat-contained connective tissues supply this buffer. When properly employed, they reduce the stress and strain of forces imposed by muscle action. The insertion of the musculature at or near the denture border influences denture stability. The character and location of these muscle attachments should be appreciated. Muscles having collagenous attachments, either tendinous or aponeurotic, are poorly suited to forming adaptive contacts owing to their lack of elasticity. The elastic quality of the supporting tissues is a prerequisite to denture retention.

Collagenous tissues form the attachment of the musculature at the posterior border of the hard palate and tuberosity of the maxilla except in an area of about 8 mm. at the midline (posterior nasal spine), where the uvula and palatopharyngeus muscles are located. The variability of the palatine structure is due chiefly to the variation in amount of fatty tissue and other characteristics in the form of the bony framework. Extreme development is responsible for the most troublesome conditions.

Treatment problems frequently occur at the lateral borders of the posterior nasal spine and in the locality of the medial boundary of the hamular notch. It is difficult to control adaptive contacts in these areas when the maxillary tuberosities and soft palate display irregularities in form and character usually accompanied by exaggerated action of the musculature.

Variations in the form and action of the tongue associated with altered conditions of the mandible and pharyngeal tissues affect the contour and development of the lingual flange of the mandibular denture. The pterygomandibular raphe, serving as the attachment of the buccinator and superior constrictor muscles, and the pharyngeal aponeurosis are delicate and very active and are adaptable to the denture mainly because of their fat-contained and glandular supporting submucosa. The tongue seems to be the primary determinant of the extent and form of the denture in its contact with the floor of the mouth. Its action affects the adaptive contacts with the prosthesis as all tissue movements at the lingual surface of the mandible and pharynx are associated and coordinated with movements of the tongue.

The facial muscles having their main insertion in the mucosa of the vestibule influence the form and adaptation of the labiobuccal denture borders. The arrangement of the facial muscles and the direction of their fibers lend themselves to denture support. The angles of the mouth form the hub of activity of the facial muscles which radiate upward, downward and laterally from this central location. All are associated with the orbicularis oris, which surrounds the orifice of the mouth, and their main structure is the buccinator muscle. Its origin in the maxilla and mandible affects both the upper and the lower denture. Its action in conjunction with that of the interdigitating muscle fibers from the superior and inferior surfaces of the face exerts a gripping force on the labiobuccal area of the dentures, contributing to their stability and retention.

The tissues of the mouth and face that form the support of prosthetic dentures deserve consideration. The success of denture service depends on their judicious employment.

Summary

The purpose of this study was to obtain a complete and exact understanding of the human anatomy of the face and mouth as related to the problem of denture prosthesis. Dissections of edentulous cadavers were prepared in sequence

from the skin of the face and mucous membrane of the mouth to the supporting bony structures. The tissues are presented from the dentist's perspective. Right, left and frontal views of the face and the mouth show the structures as they are encountered by the operator in the treatment of edentulous conditions. The accuracy of the illustrations is limited only by the limitations of technical skill.

The muscles of the facial system influence stability and retention as they affect the development of border outline and adaptation and as they engage the labiobuccal surfaces of the denture. The facial muscles of expression affect adaptive contacts by virtue of their attachments to the mucosa. The mechanism of the facial muscles of expression is complex. They may serve the stability and retention of the denture when properly employed, or they may be an impediment when encroached upon. This is true also of the musculature of the soft palate and floor of the mouth where collagenous tissues may form the attachment of the muscles to the bone.

Collagenous tissues (tendon and aponeurosis) are unsuited for use where they form adaptive contacts with the denture, because of their lack of elasticity. Fatty tissues are best suited to the contact of denture borders. Fatty and glandular tissues afford a high degree of elasticity. The denture-bearing areas of the mouth are well stocked with elastic tissues acting as buffers in the dissipation of stress and strain.

The dissections reveal the character, location and distribution of the musculature, the investing soft tissues and the form and outline of the bone structure. The understanding of the relation of these tissues to the denture problem is fundamental to the treatment of edentulous conditions.

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THE JOURNAL: PERFORMANCE AND PROMISE

The end of the first year of the present editor's tenure coincides sufficiently, for purposes of discussion, with the lifting of many wartime restrictions on paper and printing, so that the immediate past and future of THE JOURNAL may be examined with propriety. During the war, THE JOURNAL successfully met many difficult problems of production only to encounter a compositors' strike of three weeks' duration after the war had ended. In this period, THE JOURNAL maintained almost at prewar levels its volume of published material. A necessary but regrettable decline in the weight and the quality of the paper used became evident as the war proceeded, but it is hoped that this situation will improve shortly. As soon as possible, THE JOURNAL will resume its use of heavier paper stock with subsequent improvement in the ease of reading and the reproduction of illustrations.

Articles.—In order to sustain the high quality of scientific articles that are printed in THE JOURNAL, it is now an established policy to submit all manuscripts received to one or more expert consultants for evaluation. On the basis of such reports, the final editorial judgment is made. This procedure, which does increase the time needed to consider a manuscript, has been designed solely to prevent the escape into print of premature, poorly-grounded or sketchily-conceived reports that can affect the scientific standing of dentists and dentistry.

The flow of manuscripts, which one would expect to be diminished with more than one-third of all dentists in the armed services, has been little interrupted during the war. THE JOURNAL continues to receive three or four times as many manuscripts as can be printed in a given year. This fact has led to (1) an increased rejection rate so that other outlets for articles can be found without delay in state and local dental journals and (2) plans for enlarging the space available in THE JOURNAL for scientific material.

The lack of comprehensive articles on the clinical aspects of dentistry has been emphasized during the war period because, perhaps, of the pressure of military and civilian duties on all dentists. In cooperation with the Council on Dental Therapeutics, THE JOURNAL has invited outstanding clinicians to prepare a series of articles

that will be of great practical interest and value to practicing dentists. The first of this series will appear shortly.

Departments.—In an effort to provide a forum for letters of general interest to readers, for case reports and for other material that does not have the dimensions for full-fledged scientific presentation, the department "Cases and Comments" has been initiated. This will be enlarged as interest and needs dictate. The section devoted to reviews has also been expanded to permit more critical and extensive reviews of books that interest the dentist. Efforts are also being made to widen the coverage of this section so that important publications, both from here and abroad, in all fields of dentistry will receive the attention of qualified reviewers.

The end of the war has brought about the renewed exchange of periodicals with dental societies in almost every country of the world. Most of these publications still show signs of the wartime exhaustion of facilities and resources, but the material presented will become increasingly important as the countries return to their peacetime tasks. As soon as it can be arranged, THE JOURNAL hopes to present a summary of this literature from abroad in the department devoted to abstracts and summaries of current literature.

The need for an informed membership has never, perhaps, been as great as it is at the present time. Professional, technical, social, military and economic problems are under discussion everywhere. For this reason THE JOURNAL has greatly enlarged the space given to the reporting of news events in both of its issues. The activities of the various councils, committees and bureaus of the American Dental Association have also been reported in greater detail so that readers can become more familiar with their programs and their services.

Future Program.—The present plans of THE JOURNAL do not end with the changes enumerated. Many others will be made as soon as conditions permit. A reconsideration of the present format of THE JOURNAL is now underway and an increased page size to permit better presentation of articles is being given close attention. The possibility of publishing two regular issues of THE JOURNAL each month is also under study. These changes, when made, will be accompanied by a change in style and type faces to insure greater attractiveness and readability.

THE JOURNAL will continue to have as its single purpose the presentation of all types of material that will be interesting and useful to dentists. The comments of readers always serve as a valuable guide in determining the directions in which that interest and usefulness lie.

"MAN AGAINST PAIN"

For almost all of the 100 years that have elapsed since the discovery of surgical anesthesia, a considerable controversy has surrounded the names of Horace Wells, William T. G. Morton and others, all of whom possibly had some role in its discovery or presentation to the world. During the celebration of the Horace Wells Centenary in 1944, much attention was given to the re-examination of Horace Wells' real position in the controversy. It is likely that the Morton Centennial, to be celebrated in Boston this year, will occasion a similar revival of interest.

In this interval between two centennials, the publication of "Man Against Pain: The Epic of Anesthesia,"[1] written by Dr. Howard Raper, is an interesting event. In

1. Man Against Pain. New York: Prentice-Hall, Inc., 1945. See p. 253 for review.

a vivid and dramatic book, Dr. Raper tells the tale of all of the tragic figures involved in this great discovery. His views and conclusions, unlike those of many other writers on this subject, are well-documented and commendable for their objectivity. Raper finally reaches the conclusion—one that will be shared by most of his readers—that Horace Wells actually has the soundest claim but that in the immensity of this gift to humankind there is room enough for all contenders.

"Man Against Pain" may not settle this perennial discussion with finality but, certainly, its realistic appraisal of the facts cannot be neglected by any reader or historian who is interested in the fascinating history of the introduction of surgical anesthesia.

REPORTS OF COUNCILS AND COMMITTEES

COUNCIL ON DENTAL THERAPEUTICS

VI ORAL—NOT ACCEPTABLE FOR A.D.R.

This report has been authorized for publication by the Council on Dental Therapeutics. An earlier report, which was submitted to the Stuart Company, included reference to the Stuart Formula Tablets. The Stuart Company wrote on July 20, 1945: ". . . since six months ago we have not advertised The Stuart Formula to the dental profession . . . nor do we intend to do so." In view of the statement of the firm, the Council decided not to include comments with regard to the Stuart Formula in its report at this time. Advertising material received recently indicates that the name of the firm which distributes ViOral now is the ViOral Company, 234 East Colorado St., Pasadena, Calif. The product was formerly distributed by the Stuart Company at the same address.—Donald A. Wallace, *Secretary.*

ViOral is marketed in the form of tablets, each of which is stated to contain vitamin C, 100 mg.; niacinamide, 50 mg.; calcium pantothenate (dextrorotatory), 5 mg.; riboflavin, 2 mg.; and also other members of the vitamin B complex from natural sources: liver extract, yeast extract and high potency yeast, plus inert tableting ingredients: calcium pyrophosphate and tricalcium phosphate. "No sugar."

The firm states that ViOral should be used for the following "Deficiency Symptoms": "Aptha (sic), Bleeding Gums, Burning Tongue, Cheilosis, Constipation (An important contributory cause of pyorrhea), Detachment, Dry Mouth (Xerostomia), Extreme Calcium Deposits, Geographic Tongue (Glossitis), Gingivitis, Hemorrhage Tendency, Herpes, Inflamed Mucosa, Inflammation of the Lips and Tongue, Lichenplanus (sic), Loose Teeth, Pyorrhea (all forms), Recession, Ropy Saliva, Sore, Puffy Gums, Stomatitis, Slow Healing After Extraction. Vincent's Infection, Weakened Blood Capillaries."

While the appearance of some of these conditions might be associated with dietary deficiencies, such is not always the case. Recovery from dental disease would not necessarily be accelerated by the administration of vitamins and, even in those instances in which recovery might be hastened, it is unlikely that such a mixture as ViOral would be the nutritional supplement of choice.

Adequate evidence is not available to indicate that the causes of gingival hemorrhage, Vincent's infection, ulcerative gingivitis or dry sockets are confined to nutritional deficiencies, nor is there evidence that the use of ViOral will bring about firm healthy gums and decrease the tendency to post-extraction hemorrhage.

A great many dentists, as well as many physicians, are not qualified to diagnose or treat nutritional deficiency diseases. If, after proper diagnosis, it is believed that a pathological condition of the mouth is due to inadequate nutrition, the dentist, in cooperation with the patient's physician, should prescribe the diet that the patient needs. Vitamins are food supplements, not medicines. Their promiscuous administration in the treatment of dental disease is largely wasteful

and may tend to blind the patient and the dentist to the importance of the operative and surgical procedures upon which successful treatment depends.

Because of their uninformative name and the unwarranted claims made for them, ViOral Tablets are not acceptable for A.D.R.

KORAL TOOTH POWDER—NOT ACCEPTABLE FOR A.D.R.

The following report, which has been authorized for publication by the Council on Dental Therapeutics, was sent to the firm on October 30, 1945. No reply had been received from the firm at the time that this report was submitted to the Editor for publication in THE JOURNAL.—Donald A. Wallace, *Secretary.*

Since 1942, the Council office has received occasional inquiries concerning Koral Tooth Powder. The firm was given an opportunity to submit its product for consideration by the Council, but the product has not been submitted. In fact, the firm did not even reply to a registered letter, dated May 24, 1945, from the Council office.

The firm has made no secret of the fact that Koral consists of 94.5 per cent of sodium bicarbonate with minor additions of magnesium trisilicate, tribasic calcium phosphate, saccharin, soap, flavors and coloring.

However, the claims made for this product are amazing. In fact, they insult the intelligence of the dentists to whom the advertising is sent.

Examples of the claims made are:

Koral Tooth Powder neutralizes mouth acids and maintains alkalinity twice as long as milk of magnesia.

The implication is that the product is useful in preventing or retarding dental caries. Neither sodium bicarbonate nor milk of magnesia has been shown to be effective in preventing or retarding caries.

. . . dentifrices which fail to neutralize local acid conditions are worse than none at all. . . .

Again the misleading inference is that the use of alkaline dentifrices will prevent dental caries.

In post-operative treatment of Gingivitis, Koral is of very great value. Its effectiveness in augmenting the treatment of Gingivitis lies in the high percentage of bicarbonate of soda in its composition.

So far as the Council members are aware, sodium bicarbonate does not have the remarkable virtues attributed to it in this statement.

Other misleading claims, some even more objectionable than those cited, have been made for the product in the past. However, claims change with the times and the firm may think of others which it has not yet used. It is essential to remember that, while a proper grade of sodium bicarbonate may be a good dentifrice, currently available information indicates that it can do no more than help the toothbrush clean the accessible surfaces of the teeth.

To summarize: Koral Tooth Powder is essentially sodium bicarbonate. The unwarranted and exaggerated therapeutic claims made for it are against the interests of the public and of the dental profession. It is therefore declared unacceptable for A.D.R.

NOTICES OF JUDGMENT

1255. Adulteration and misbranding of procaine hydrochloride solution, with epinephrine. U. S. v. 38 Packages of Procaine Hydrochloride Solution (and 3 other seizure

actions against procaine hydrochloride solution). Default decrees of condemnation and destruction. (F.D.C. Nos. 12348, 12407, 12509, 12774. Sample Nos. 35967-F, 35968-F, 50975-F, 63447-F, 75324-F, 75349-F.)

Between the approximate dates of May 10 and June 28, 1944, the United States attorneys for the Northern District of Georgia, the Eastern District of Pennsylvania and the Northern District of Ohio filed libels against the following amounts of procaine hydrochloride solution: Fifty-two packages, each containing twenty-five cartridges, at Atlanta, Ga.; thirty-eight packages, each containing twenty-five cartridges, at Philadelphia, Pa., and 200 cartridges at Youngstown, Ohio; alleging that the article had been shipped between the approximate dates of March 8 and May 15, 1944, by A. Pfingst and Pfingst & Co., New York, N. Y., and charging that it was adulterated and misbranded. The article was labeled in part: "Procaine Hydrochloride (or "HC1") Solution 2% with Epinephrine."

The article was alleged to be adulterated in that its purity and quality fell below that which it purported to possess since the article was not sterile, but was contaminated with living micro-organisms.

The article was alleged to be misbranded in that it was dangerous to health when used in the dosage suggested in the labeling thereof; that is, when the contents of the cartridge were injected into the tissues. A portion of the article was alleged to be further misbranded in that it failed to bear a label containing an accurate statement of the quantity of the contents of the package. The label of this portion bore no statement of the quantity of the contents of each cartridge.

Between May 29 and July 26, 1944, no claimant having appeared, judgments of condemnation were entered and the product was ordered destroyed.

The formerly accepted dosage forms of the Pfingst local anesthetic solutions were deleted from Accepted Dental Remedies on August 17, 1943.

1300. Misbranding of an unlabeled drug product. U. S. v. 768 Packages of an Unlabeled Drug Product. Decree of condemnation. Product ordered released under bond. (F.D.C. No. 10952. Sample No. 3924-F.)

On or about October 20, 1943, the United States attorney for the Western District of Missouri filed a libel against 768 packages of an unlabeled drug product at Kansas City, Mo., alleging that the article had been shipped on or about September 14, 1943, from Detroit, Mich., by the Nu-Basic Products Co.

Analysis of a sample showed that the article consisted essentially of unsaponifiable oil containing small proportions of carbolic acid, sulfanilamide, a saponifiable oil, and water.

The article was alleged to be misbranded in that it did not bear a label containing the name and place of business of the manufacturer or distributor, nor an accurate statement of the quantity of contents in terms of weight or measure; and in that it was fabricated from two or more ingredients and was not labeled to show the common or usual names of the active ingredients.

On October 26, 1943, the Nu-Basic Products Co. having appeared as claimant, judgment of condemnation was entered and the product was ordered released under bond for labeling in compliance with the law, under the supervision of the Food and Drug Administration.

COUNCIL ON DENTAL HEALTH

SUMMARY OF THE NEW WAGNER-MURRAY-DINGELL BILL

President Truman announced his proposals for a national health program in a message to Congress November 19, 1945. A new Wagner-Murray-Dingell Bill was introduced immediately in both houses, S. 1606 and H.R. 4730. There are now two different Wagner-Murray-Dingell bills in Congress, but it is presumed that the last one, S. 1606, replaces S. 1050.

Some major changes were made in the new bill, probably for reasons of strategy. For example, the provisions for collecting contributions from employers, employes

and self-employed persons were eliminated completely. This was done so that the bill could be referred to the Committee on Education and Labor, of which Senator Murray is chairman, and thus receive a favorable hearing in committee. Previous Wagner-Murray-Dingell bills were referred to the Senate Committee on Finance and were never reported out of committee. It seems obvious that a new bill or amendments to the present bill will need to be introduced to make provisions for compulsory contributions. Such amendments will probably be made at a later date.

A large portion of the first section was deleted completely. This section was almost identical with the provisions of the Hill-Burton Hospital and Health Center Construction Act. The reasons for removing this section are not entirely clear, but one reason might be the fact that the Hill-Burton Bill is slated for passage; in fact, it has been adopted by the Senate. It is logical to assume that the authors would want to delete the provisions that will probably be adopted by Congress in another measure. A brief summary of the more important sections in the new bill follows:

1. Grants to states, counties and health districts: (*a*) for the prevention, treatment and control of venereal disease and tuberculosis and (*b*) for the establishment and maintenance of adequate public health services.

2. Grants to the states for maternal and child health services for "all mothers and children in the state or locality who elect to participate in the program."

3. Appropriations to enable each state to provide services for "all crippled children . . . who elect to participate."

4. Grants to states to provide medical care for needy persons.

5. Prepaid personal health service benefits (general medical, special medical, general dental, special dental, home nursing, laboratory and hospitalization benefits). Almost all persons would be entitled to receive personal health service benefits.

a. The Surgeon General is directed to negotiate agreements with agencies, institutions, groups and individuals "to utilize their services and facilities and to pay fair, reasonable and equitable compensation for such services and facilities." (The Surgeon General can delegate this responsibility to others.)

b. The Surgeon General shall appoint local committees to aid in the administration of the Act. The members of such committees shall be selected from panels of names submitted by the professional organizations concerned with medical, dental, nursing, hospital and laboratory services.

c. A national advisory medical policy council would be appointed and would consist of the Surgeon General as chairman and sixteen members selected from panels of names submitted by professional and other organizations concerned with medical, dental, nursing, hospital and laboratory services.

d. Any dentist or physician may elect to participate in the plan and may select a method of payment based on salary, fee for service, per capita or a combination of these methods.

e. Reimbursement for medical care for the needy would be provided under the same methods of payment to practitioners.

f. The Surgeon General may, after consultation with the Advisory Council and with the approval of the Administrator, determine for any calendar year or part thereof that every individual entitled to health services may be required by the physician, dentist or nurse to pay a fee with respect to the first service, or with respect to each service in a period of sickness or course of treatment. Such determination may also limit the application of such fee to home calls or office visits, and may fix the maximum total amount of such fee payments and may also provide for differences in the maximum size of such fees for urban and rural areas and with regard for differences among states and communities. (It is believed that this clause was inserted to control abuses or malingering.)

The Surgeon General, having regard for the adequacy of available personnel, may, after consultation with the Advisory Council and with the approval of the Administrator, determine for any calendar year that dental benefits shall be restricted to such services as the Surgeon General may determine, provided the restricted services include at least examina-

tion (including x-ray survey) and diagnosis; prophylaxis; extraction of teeth which are considered by the dentist and an attending physician to be or likely to be injurious to the general health of the individual; and treatment of acute diseases of the teeth, their supporting structures and fractures of the teeth or jaws. (It is not clear if carious lesions in teeth would be considered to be acute diseases of the teeth.)

With respect to general dental or special dental benefits, such determination may fix an age above which the restrictions on content shall apply. (It is important to note that the restriction in dental service could be limited to older age groups, thus making children eligible to receive complete service.)

g. The Act would create on the books of the U. S. Treasury a separate account to be known as "Personal Health Service Account." The Act would also authorize the appropriation to the account of such sums as may be required to finance the benefits, payments and reimbursements. From such appropriations, the Secretary of the Treasury would credit to the account an amount equal to 3 per cent of the wages paid after June 30, 1946, with respect to employment.

From such appropriations, the Secretary of the Treasury would credit annually to the account amounts estimated by the Surgeon General with respect to the preceding fiscal year to have been expended for the payment or provision of health care.

h. Provisions are made to finance medical education and research. Ten million dollars would be made available for the year 1946, fifteen million dollars for the year 1947 and for each calendar year thereafter an amount equal to 2 per cent of the amount expended for health care.

The bill contains many qualifying phrases, making it very difficult to read. It also postpones important decisions on administrative detail until after the bill is enacted into law. It is therefore difficult, if not impossible, to determine how some of the provisions would be carried out and what would be the functions of federal and state agencies in administering the prepaid personal health service.—*Allen O. Gruebbel, D.D.S., Executive Secretary.*

COMMITTEE ON LEGISLATION

SUPREME COURT UPHOLDS "MAIL-ORDER DENTURE" LAW

The United States Supreme Court, on October 8, 1945, refused to hear an appeal from the United States Circuit Court of Appeals involving the constitutionality of the Traynor Law. This action means that the appellate court decision upholding the constitutionality of the Traynor Law stands as the final decision in the case. Thus, the Federal Denture Act, more commonly known as the Traynor Law, in honor of Philip A. Traynor, of Delaware, a dentist, who introduced the bill in Congress, has now been determined by the courts to be constitutional. The last case to be taken to the courts involved the United States Dental Company, which claims to be the world's largest maker of dentures, and which has advertised extensively throughout the country.

At the trial in the United States District Court, held in September 1944, the defendants were found guilty and an appeal was taken to the United States Circuit Court of Appeals, where the judgment of the lower court was upheld in a decision stating that the Traynor Law was clearly constitutional. This decision, made on April 27, 1945, was reported in the June 1, 1945 issue of THE JOURNAL, page 764.

An appeal was then taken to the

United States Supreme Court and, on October 8, 1945, the court issued an order refusing to hear the appeal. This means that the decision of the United States Circuit Court of Appeals became the final decision in the case, and it is important to remember that, in its decision, the Circuit Court held that "such legislation is constitutionally unchallengeable."

A history of the mail-order denture problem is contained in a report of the Committee on Legislation in the October 1, 1945 issue of THE JOURNAL, pages 1305-1306. Briefly, the record reveals that prior to the enactment of the Traynor Law, the United States Postoffice Department sought to protect the public through the use of fraud orders which would deny mail-order dental laboratories the right to use the mails. The use of fraud orders was only partially successful, presenting many difficult problems that interfered with the efficiency and success of the orders.

It was realized that the problem must ultimately be solved by the enactment of a federal law, prohibiting the use of the mails or any other instrumentality of interstate commerce. As a result, Congressman Traynor introduced a bill which finally was enacted on December 24, 1942, and came to be known as the "Federal Denture Act," or as the "Traynor Law."

Over a period of three years thereafter, three test cases were taken into the courts, two of which went to the United States Supreme Court.

When a law passed by Congress is attacked on the ground that it is unconstitutional, the Justice Department usually withholds universal enforcement of the law and takes one or more cases, commonly known or referred to as test cases, through to the United States Supreme Court, in order to secure the decision of the highest tribunal before further prosecutions are undertaken.

As regards the Traynor Law, two test cases were taken to the United States Supreme Court, and it is now believed that all questions concerning its constitutionality and the proper methods for enforcing it have been determined.

From now on, there should be no more violations of the Traynor Law. Hence, any member of the profession observing a violation in the future should report it immediately to the Committee on Legislation, American Dental Association, 222 East Superior St., Chicago 11, Ill.

NEWS OF DENTISTRY

Full Text of Board of Trustees' Resolution on Changes at Columbia

Following is the full text of the resolution approved by the Board of Trustees of the American Dental Association in October. The resolution deals with the conversion of the Columbia University School of Dental and Oral Surgery into a dental department of its Faculty of Medicine:

The Board of Trustees of the American Dental Association notes with great regret that, as of July 1, 1945, Columbia University converted its School of Dental and Oral Surgery into a dental department of its Faculty of Medicine.

The Board has been informed also that, long before final action was taken by the University, a majority of the members of the Faculty of Dentistry voted against the proposed subordination of dental education in that University; and that the Dental Alumni Association of Columbia University, and the Dental Societies of the State of New York

and of the First and Second Districts (Greater New York City), presented to the Columbia University Board of Trustees formal expressions of opinions that the School of Dental and Oral Surgery should be continued under its own faculty on a par with all other professional schools in the University.

The authorities at Columbia have not made clear the advantages for the public, for dental education or for the dental profession, in this subordination of dentistry, which would not result from the continuance of the School of Dental and Oral Surgery in appropriate coordinations with the other health-service and science schools and departments in the University.

That the said action by Columbia University was not due to inefficiency of the Dental Faculty is indicated by the facts that the Council on Dental Education of the American Dental Association had recently rated highly the School of Dental and Oral Surgery; had reported privately to representatives of the University that the school was doing excellent work; and, in the Council's public report published last June on the status of all dental schools in this country, included the School of Dental and Oral Surgery in the group of approved schools.

Available copies of records of the proceedings of the Faculty of the School of Dental and Oral Surgery indicate that the subordination of dental education to medical education at Columbia University was proposed by the Dean of the Medical School, who has held that position since 1931, and who, since 1933, has also been Dean of the Dental School. His longstanding feeling on medico-dental relationships is indicated by such published statements as these:

(*a*) In 1932, the "Final Report of the Commission on Medical Education," issued by him as "Director of Study" from his office as Dean of the Medical School of Columbia University, contained on page 217 this advisory recommendation: "Dentistry should be developed *under* medical education."

(*b*) In 1933, his Annual Report as Dean of the School of Dental and Oral Surgery of Columbia University (pamphlet edition, page 7) contained this gross misstatement: "Oral surgery, *of course*, is *already* recognized as a specialty of *medical* practice."

The decision at Columbia University to subordinate dental education to medical education, apparently as a prelude to efforts to dismember dentistry, raises a variety of questions, among them: Should dentistry be discontinued as an autonomous profession? And if so, why? The correct answers to these questions are grounded in promotion of the public welfare and are rooted in the history of dentistry and of dental education, some of the basic facts of which are these:

(1) Dental practice has existed as a natural division of health service from the earliest accounts in history.

(2) The form and the structure of dental practice have been developed effectually down through the centuries in response to social needs requiring biochemical methods of therapeutic treatment that conventional medicine, traditionally unconcerned about dental diseases and their consequences, never provided.

(3) The autonomous system of institutional dental education, initiated in the United States over 100 years ago, has advanced dental health service in this country to universally accredited world pre-eminence in quality—including all countries in which dental education is dominated by medical education.

(4) During the past century, the autonomous dental profession, because of its concern, understanding, fidelity and successful efforts—unhampered by indifferent medical direction—has acquired special competence in the related sciences and arts of dental health care, the special knowledge and skill of which those who select medical practice as their profession usually do not possess, do not need and do not acquire.

(5) The natural evolution of dentistry independent of medicine—in full accord with the prevailing preferences of both professions—has resulted in the establishment of a separate system of education, the growth of a special literature and the development of a complex pattern of local, state and national organizations, and has thus created a closely knit professional group that is duly accredited and regulated by special statutes in all of the states, the number of active licensed dentists being approximately equal to half the number of general and special medical practitioners combined.

(6) A decision to obstruct or change the course along which dental education and dental practice have been proceeding in the

United States during the past century—by placing them under medical domination—would lower dentistry in quality, in usefulness, in public esteem and in public confidence. Such a decision would also be based on the unwarranted assumption that dental health service would be made more efficient by a profession that has been traditionally inattentive to dental disorders than it would be by the sustained initiative, understanding and concern of the competent, well organized and respected profession that has shown itself to be able not only to evolve dental health care to its present excellence but also expertly to continue that development in the public interest.

What the medical profession thinks of such intrusion into its own professional responsibilities—and also what it presumably concedes in principle as to dentistry's professional responsibilities—was stated clearly on page 36 of "Medical Education in the United States in 1934-1939," issued by the Council on Medical Education and Hospitals of the American Medical Association:

"Whatever the administrative organization might be in any particular school, it would seem to be *educationally and professionally unsound* to entrust the responsibility for the development of students in medical fields to others than medically qualified teachers. The *safeguarding of the medical point* of view can be entrusted in the universities as they exist in this country *only to those interested and competent in medical education.*" (Italics not in the original.)

Therefore, with the foregoing and other kindred facts and conditions under consideration, be it

Resolved, that in the judgment of the Board of Trustees of the American Dental Association, the autonomous development of dentistry as a natural division of health service is an essential condition for the best possible dental health care in this country in the future as it has been during the past; and also

Resolved, that the evolution of the best possible dental health care for all the people requires continuance and furtherance of intimate cooperation between individual physicians and dentists in practice and between medical and dental faculties in university professional education; with due recognition of the facts that the medical and dental professions are independent, that their health services should be informally interdependent, and that their personal and professional relationships should be mutually expressive of the ideal of coordination without subordination; and be it also

Resolved, that this Board declares its firm opposition to any movement in dental education that reduces or tends to lower dental education in rank and authority in the university program; and denounces any existing situation or plan in dental education or practice that restricts the freedom of initiative and enterprise that dental leadership has exercised heretofore, or that subjects the direction and control of dental functions to domination by a faculty of medicine or of other medical authority; and be it further

Resolved, that these resolutions be sent to the following: the Council on Dental Education, the secretaries of all component societies of the American Dental Association, the editors of all state dental journals, the deans of all dental schools, the deans of all medical schools, the Board of Regents of the State of New York, the president of Columbia University and the presidents of all universities that have medical and dental schools; and be it further

Resolved, that these resolutions be presented to the House of Delegates of the American Dental Association with a recommendation from the Board of Trustees for their adoption at its next regular meeting.

New York State Board Opposes Subordination of Dental Education

At the annual meeting of the New York State Board of Dental Examiners, December 4, Dr. Waldo H. Mork, president of the Dental Society of the State of New York, condemned the action of the Trustees of Columbia University in merging the dental college with the medical school.

Irwin A. Conroe, assistant commissioner for professional education of the New York State Education Department, stated at the meeting that no action would be taken to change the status of the Columbia University dental school

until such time as there was evidence that the standard of the dental teaching and practical instruction was lowered.

It was stated by the New York State Board of Dental Examiners that any plans or means of education which would tend to subordinate the dental profession to any of the other professions would be opposed. It was also proposed at the meeting that deans of all dental colleges be graduate dentists.

Project to Study Dental Needs of Children Started in Rhode Island

The U. S. Public Health Service, in cooperation with the Rhode Island State Health Department, is conducting a dental care demonstration in Woonsocket, R. I., to determine the annual incidence of dental needs in school children and to obtain first-hand information on the professional services required to care for those needs.

Approximately 7,000 school children are eligible for dental care under the program. All dental services, except orthodontics, are rendered in clinics set up in the school buildings. Each clinic is equipped with the standard equipment required to conduct a modern pedodontic practice.

Six dental officers of the Public Health Service have been assigned to the project. A sufficient number will be assigned to care for the current dental needs of the children in a period of two to three years. Thereafter, the number of dentists will be adjusted to the maintenance needs of the children. It is anticipated that the objectives of the study will be attained in five to six years.

Each dentist is provided with two complete dental units and is assisted by a trained dental assistant. Dental hygienists perform all prophylaxis, and clerical personnel assist in maintaining complete dental records.

Consulting pedodontists will be engaged at intervals to instruct the operators in modern technics and practices in children's dentistry. In addition, consultants will be employed to assist the dentists in acquiring ability to utilize dental assistants effectively.

The city of Woonsocket, with a population of approximately 50,000, is contributing 30 per cent of the cost of operating the project, and the Rhode Island State Department of Health will contribute 30 per cent of the cost beginning July 1, 1946.

State and local dental societies officially approved the study prior to its inauguration in December 1945. The program is the third dental demonstration set up by the Public Health Service during the past year.

116 Students and Educators Attend Akron Dental Society Banquet

The Akron Dental Society's student-dentist banquet, held December 10, was attended by 116 student guests as well as educators from nearby schools and colleges. The banquet was held to interest high school boys and girls, college students and veterans in the advantages of dentistry as a career. The society reports that the need for dentists is at its highest point since before the war.

Among the speakers were Dean W. L. Wylie, Western Reserve University, School of Dentistry, who spoke on "Opportunities in Dentistry," and Dean Wendell D. Postle, Ohio State University, College of Dentistry, who discussed "Pre-Dental Curriculum."

National Social Hygiene Day to Be Held February 6

The American Social Hygiene Association announced recently that its National Social Hygiene Day will be held February 6. Since 1914, the meeting has been attended by officials and citizens from all parts of the country to consider local, state and national social hygiene problems.

War Service Committee to Hold Open Meeting February 9

The War Service and Postwar Planning Committee of the American Dental Association announced recently that it will hold an open session in cooperation with the Chicago Dental Society at 9:30 a.m., February 9, at the Stevens Hotel, Chicago, Ill.

C. Willard Camalier, chairman of the committee, will preside at the meeting. The morning program will open at 9:30 with an address given by Walter H. Scherer, President of the American Dental Association. At 10:00 Maj. Gen. Robert H. Mills, Chief of the Dental Division of the U. S. Army, will talk on "Demobilization of Dental Officers," after which C. W. Freeman, chairman of the Postwar Planning Committee of the Illinois State Dental Society, will discuss "Dental Officer Returns to Civilian Practice—Problems and Remedies."

The noon recess will be followed by an address by Sterling V. Mead, President Elect of the American Dental Association. At 2:00 Watson B. Miller, Federal Security Administrator, will discuss the subject "Surplus Dental Materiel." At 3:00 Allen O. Gruebbel, chairman of the Veterans Administration Program Committee of the American Dental Association, will introduce Milburn M. Fowler, Chief of the Dental Division of the Veterans Administration, who will talk on "Dental Care Program of the Veterans Administration." The last event of the day will be a discussion of a "Memorial for Dental Officers," at which time suggestions for the type of memorial and recommendations for future action will be made.

All members of the dental profession are invited to be present to enter the discussions, propound inquiries and present resolutions on any of the subjects embodied in the agenda.

State Revokes G. I. Aid for National School of Mechanical Dentistry

As the result of an examination taken upon the complaint of fifteen students of the National School of Mechanical Dentistry in Chicago, Vernon L. Nickell, state superintendent of instruction, has halted payment of G. I. benefits to veterans attending the school, it was announced recently.

The Illinois State Dental Advisory Board found the staff and physical facilities of the school inadequate and held that graduates were not likely to find employment. Charges were denied by S. T. Jonas, head of the school. The majority of the 100 students attending the school are veterans being trained as technicians for dental laboratories.

Public Health Dentists Appoint New Officers

The new officers and Executive Council members announced by the American Association of Public Health Dentists are: Frank C. Cady, president; Edward Taylor, president-elect; John W. Knutson, Executive Council; Olin E. Hoffman, Executive Council.

New York Dental Hygienists to Hold Meeting February 1

The Dental Hygienists Association of New York City will hold its regular monthly meeting at 8 p.m., February 1, at the Hotel Pennsylvania, New York.

Washington University Dental Alumni to Hold Meeting February 21-22

The annual meeting of the Washington University Dental Alumni will be held in the Washington University, School of Dentistry, February 21-22.

LIST OF ARMY DENTAL OFFICERS SEPARATED FROM SERVICE

The following dental officers have been separated from service, according to lists received from the Dental Division, Office of the Surgeon General, U. S. Army. These lists are not complete. Publication will be given to other names as soon as possible. Lists for separated naval dental officers have not been available.

	Alabama	
Andalusia	McCarty, William L.	Maj.
Birmingham	Hollister, John F.	Maj.
Birmingham	Rainwater, Lem H.	Capt.
Cuba	Walker, Douglas E.	Capt.
Ensley	Bobo, Herbert O.	Capt.
Gadsden	Leach, James E., Jr.	Capt.
Jacksonville	Beard, Andrew J., Jr.	Maj.
Opelika	Hagood, M. H., Jr.	Maj.
	Arizona	
Jerome	McMillan, Ralph W.	Maj.
	Arkansas	
Hope	Hardage, Albert L.	Lieut.
Hot Springs	Turk, Raymond C.	Lt. Col.
	California	
Alameda	Caplan, Charles C.	Capt.
Alhambra	Fink, Edwin H.	Lt. Col.
Auburn	Cotton, Norman W.	Capt.
Bakersfield	Cuneo, George H.	Capt.
Berkeley	McKimmins, Mark	Maj.
Berkeley	Nordstrom, Herbert L.	Maj.
Berkeley	Salera, Dante	Capt.
Beverly Hills	Maimes, Robert M.	Capt.
Downey	Sims, John C.	Capt.
Fresno	Dau, Chester J.	Capt.
Fresno	Guthrie, Paul R.	Lieut.
Goleta	Hobbs, Angus G.	Capt.
Los Angeles	Gordon, Daniel F.	Lieut.
Los Angeles	Griffing, Burell E.	Capt.
Los Angeles	Singleton, Lawrence S.	Maj.
Los Angeles	Williams, Charles R.	Capt.
Modesto	Leonard, Fred J.	Capt.
Monterey Park	Birk, Arthur F.	Capt.
Northridge	Fink, Theodore H.	Capt.
Oakland	Brown, Lloyd F.	Lieut.
Oakland	De Feo, Louis F.	Capt.
Oakland	Fong, Chester	Capt.
Oakland	Schafer, Orrin H.	Capt.
Oakland	Wilson, Kermit A.	Capt.
Pasadena	Gillam, Claude E.	Capt.
Pasadena	Kruell, Herman A.	Capt.
Redwood City	Burns, Stanton R.	Capt.
Sacramento	Stark, Verne A.	Maj.
Sacramento	Sylva, Earle A.	Maj.
San Diego	McAmis, Leon C.	Capt.
San Francisco	Charyn, Harold C.	Capt.
San Francisco	Frank, Roy M.	Capt.
San Francisco	Giacalone, Anthony J.	Capt.
San Francisco	McGann, William R.	Capt.
San Francisco	Seagrave, Charles E.	Capt.
San Francisco	Suppo, Andre D.	Maj.
San Francisco	Susnow, Samuel I.	Capt.
San Francisco	Zucca, Alfred J.	Capt.
San Jose	Breene, Frank E.	Maj.
San Mateo	Guy, Lewis H.	Maj.
Taft	Dienstein, Benjamin	Capt.
Woodland	Fisher, Daniel E.	Capt.
	Colorado	
Denver	Bishop, Donald K.	Capt.
Denver	Carlin, William P.	Capt.
Denver	Metzger, Arthur B.	Maj.
Ft. Collins	Warner, Harold C.	Capt.
La Junta	Rowden, Fred A.	Capt.
Longmont	Clark, Thomas O.	Capt.
Paonia	Bradshaw, Louis C.	Capt.
Pueblo	Kellog, Howard D.	Capt.
Springs	Sinton, James J.	Maj.
	Connecticut	
Banfor	Schwartzmann, Walter	Capt.
Bethel	Moore, Allen H.	Capt.
Bridgeport	Rheinfeld, Paul J.	Capt.
Hartford	Garacciolo, P. R.	Capt.
Hartford	Shea, Erwin E.	Capt.
Hartford	Weinstein, David H.	Capt.
New Canaan	Groher, Samuel	Capt.
New Haven	Allinson, Sydney M.	Capt.
New Haven	Faulkner, Newton E.	Lieut.
Plantsville	Hurle, Robert T.	Capt.
Shelton	Steinman, Herbert M.	Capt.
Stamford	Rosenblum, Elmer M.	Capt.
Waterbury	Lawlor, James P.	Capt.
West Hartford	Taute, Rudolph J.	Capt.
Willimantic	Fox, Harry	Capt.
	Delaware	
Wilmington	Klein, Murray M.	Lieut.
Wilmington	Levy, Leon	Capt.
	District of Columbia	
Washington	Cohen, David	Capt.
Washington	Gilligan, Raymond A.	Capt.
Washington	Lane, David P.	Capt.
Washington	Leishear, Samuel A.	Maj.
Washington	Lewis, Clarence	Capt.
Washington	Neaverth, Elmer J.	Maj.
Washington	Woronow, Albert E.	Capt.
	Florida	
Graceville	Williams, Neal B.	Maj.
Lakeland	Barnes, Ward L.	Capt.
Miami	Bosworth, Oliver B.	Capt.
Miami	Frank, Lester H.	Capt.
Miami	Renedo, Henry, Jr.	Capt.
Panama City	Kramer, Abraham F.	Lieut.
St. Petersburg	Cummings, Paul M.	Maj.
Tampa	Demmi, Stephen F.	Capt.

Tampa	Jones, Thomas K.	Capt.
Tampa	Schwartz, Julius L.	Capt.
Vero Beach	Damerow, Carl F.	Lieut.
Winter Park	Gager, Walter O.	Capt.
	Georgia	
Atlanta	Hewitt, Lewis M.	Capt.
	Idaho	
Idaho Falls	Lee, Dale L.	Capt.
Ketchum	Supple, Francis J.	Capt.
Montpelier	Meek, Donald N.	Capt.
	Illinois	
Amboy	Curtin, Richard E.	Capt.
Avon	Gordon, Robert M.	Lieut.
Batavia	Nelson, Frank A.	Capt.
Brookfield	Grundset, Kenneth W.	Capt.
Camp Grant	Schulz, William A.	Capt.
Champaign	Daily, Joe W.	Capt.
Chicago	Berk, Dayton B.	Lieut.
Chicago	Berman, Harry	Lieut.
Chicago	Binotti, Evo J.	Capt.
Chicago	Bobalek, Stephen L.	Capt.
Chicago	Buckley, Joseph B.	Lieut.
Chicago	Cassidy, Thomas J.	Capt.
Chicago	Curshan, Martin S.	Capt.
Chicago	Debski, Henry T.	Capt.
Chicago	Deplewski, Matthew R.	Maj.
Chicago	Dittmar, G. W., Jr.	Capt.
Chicago	Dullaghan, Owen T.	Capt.
Chicago	Ebin, Milton J.	Capt.
Chicago	Ferencz, Albert	Capt.
Chicago	Gault, Howard H.	Capt.
Chicago	Goode, Louis A.	Capt.
Chicago	Gould, Jules S.	Capt.
Chicago	Horwitz, Henry	Maj.
Chicago	Hurwich, Ezra I.	Capt.
Chicago	Imber, Elmer	Lieut.
Chicago	Kaiser, William J.	Capt.
Chicago	La Vere, Frank C.	Capt.
Chicago	Libbin, Samuel S.	Capt.
Chicago	Manning, John L.	Capt.
Chicago	Matousek, George J.	Capt.
Chicago	Maurice, Charles G.	Capt.
Chicago	Mintz, Harold S.	Capt.
Chicago	Olech, Ray A.	Lieut.
Chicago	Poronsky, John A.	Capt.
Chicago	Salzberg, Leonard B.	Capt.
Chicago	Scranton, Richard V.	Capt.
Chicago	Sherwin, Leonard J.	Capt.
Chicago	Shrago, Richard	Lieut.
Chicago	Siedlinski, Valentine E.	Maj.
Chicago	Trace, Lester	Capt.
Chicago	Wallace, Seward C.	Capt.
Chicago	Weinshenker, Toby	Capt.
Chicago	Weller, George R.	Capt.
Evanston	Barnhart, F. P., Jr.	Lt. Col.
Farmer City	Stoughton, Frank R.	Capt.
Farmington	Wood, Arthur C.	Lt. Col.
Freeport	Griffo, Peter P.	Capt.
Joliet	Pokorny, Joseph W.	Capt.
Lawrenceville	Armstrong, Russell H.	Capt.
McHenry	Murphy, L. B., Jr.	Capt.
Madison	Wiseman, James A.	Lieut.
Marion	Judge, Newell I.	Capt.
Mattoon	Podesta, William L.	Maj.
Millstadt	Holdener, Vincent C.	Lieut.
Morris	Graham, Fred W.	Capt.
Mount Olive	Droste, Harold N.	Capt.
Murphysboro	Ozburn, Glenn W.	Capt.
Peoria	Watson, Karl J.	Lieut.
Pinckneyville	Kane, Ralph L.	Capt.
River Forest	Ulvestad, G. E., Jr.	Capt.
Rock Island	Helpenstell, Fred M.	Maj.
Rock Island	Krueger, Arnold E.	Capt.
Staunton	McBrien, Frederick R.	Capt.
Sycamore	Hennis, Harry W.	Maj.
Sycamore	Newman, Russell E.	Capt.
Vienna	Hundley, Royce S.	Capt.
Waterloo	McConnell, Justin P.	Capt.
Wood River	Roberson, George V.	Lieut.
Woodstock	Eckert, Elmer A.	Capt.
	Indiana	
Austin	Judd, Marvin E.	Maj.
Bicknell	Vendes, Walter H.	Capt.
Bloomfield	Heidenreich, F.	Capt.
Evansville	Lieberman, H. G.	Capt.
Evansville	Milbanks, John H.	Capt.
Ft. Wayne	Goshert, Joe L.	Capt.
Ft. Wayne	Scheff, Lawrence J.	Capt.
Indianapolis	Eads, Lepel C.	Capt.
Indianapolis	Fisher, Benjamin J.	Maj.
Indianapolis	Goode, Frank O.	Capt.
Indianapolis	Hanning, William F.	Lieut.
Indianapolis	Kunkel, Lucas F.	Maj.
Indianapolis	Spector, Louis D.	Capt.
Indianapolis	Unger, Edward H.	Capt.
Kokomo	Naftzger, Robert E.	Capt.
Mentone	Van Gilder, Donald J.	Capt.
Michigan City	Vojak, Cyril John	Capt.
Milltown	Lemmon, Nicholas R.	Capt.
Muncie	Wasson, Clinton I.	Maj.
Rockville	Crockett, Harold M.	Maj.
Terre Haute	Blackwell, Floyd H.	Capt.
Terre Haute	Fodora, Albert A.	Capt.
Terre Haute	Madden, R. H.	Lt. Col.
Terre Haute	Rutledge, Guy B.	Capt.
	Iowa	
Cedar Rapids	Naibert, Arthur C.	Capt.
Dayton	Ferguson, Frank H.	Maj.
Des Moines	Hemminger, John B.	Maj.
Des Moines	Rinkler, Samuel F.	Maj.
Dubuque	Plass, Forrest T., Jr.	Capt.
Guttenberg	Nemmers, Clarence J.	Capt.
Humboldt	Williams, Paul W.	Capt.
Iowa City	Easton, George S.	Lt. Col.
Lake View	Kestler, Meredith W.	Capt.
Manning	Kelsey, Raymond B.	Capt.
Mondamin	Klutts, Harlan F.	Maj.
Spencer	Kent, Robert C.	Capt.
Webster City	Oosterhuis, A. M.	Lieut.
Woodward	Baker, George H.	Capt.
	Kansas	
Atchison	Morley, Aloysius R.	Capt.
Herington	Pierce, Russell F.	Capt.
Wichita	Schubert, A. W. G.	Capt.
	Kentucky	
Corbin	Ballou, Orville L.	Capt.
Covington	Mueller, William L.	Capt.
Dawson Springs	Hawton, Floyd D.	Maj.

Harrodsburg	Flynn, Glenn G.	Capt.
Lexington	Hoffman, George S.	Maj.
Lexington	Kesheimer, Julian F.	Capt.
Lexington	West, Robert S.	Maj.
Louisville	Clements, William F.	Maj.
Louisville	Hahn, Edward J.	Maj.
Mt. Olivet	McGinety, William E.	Lieut.
Owensboro	Stringer, Peter B.	Capt.
Pikeville	Hatcher, Walter T.	Capt.
Russellville	Snider, Oscar B.	Maj.
Summer Shade	Bowman, Clarence G.	Capt.

Louisiana

Baton Rouge	Peters, William V.	Capt.
Bossier City	Sanderson, Wright W.	Capt.
La Place	Ory, Oscar J., Jr.	Capt.
New Orleans	Britsch, Louis D.	Capt.
New Orleans	Carey, Mark O., Jr.	Capt.
New Orleans	Elmer, Leonard J.	Capt.
New Orleans	Flinn, James A., Jr.	Capt.
New Orleans	Fransen, Raphael R.	Lieut.
New Orleans	Laufer, Morris H.	Capt.
New Orleans	Marquer, Victor B.	Maj.
New Orleans	Peterson, Harold W.	Capt.
New Orleans	Rossner, Charles W.	Col.
New Orleans	Sunseri, Philip D.	Capt.
Port Sulphur	Schwartz, Louis S.	Capt.
Ville Platte	Le Bas, Robert H.	Capt.
West Monroe	Gilliland, James W.	Lieut.

Maine

Houlton	Carr, Wilder D.	Maj.
Portland	Pollard, Henry	Capt.

Maryland

Baltimore	Badger, Walter L.	Maj.
Baltimore	Castelle, Paul B.	Lieut.
Baltimore	Cohen, Sigmund	Capt.
Baltimore	Dubansky, Paul S.	Capt.
Baltimore	Freedman, Gerson A.	Capt.
Baltimore	Inman, Byron W.	Capt.
Baltimore	Johnston, H. L.	Lt. Col.
Baltimore	Slavinsky, Edwin A.	Capt.
Cambridge	Smith, Wallace P.	Maj.
Lawson	Seidler, Alanzo L.	Maj.
Poolesville	Cruit, Edwin D.	Capt.
Silver Spring	Kerans, Edwin C.	Capt.

Massachusetts

Belmont	Barry, Edward H.	Capt.
Brighton	Gold, Milton	Capt.
Brighton	Lubin, Bertram	Capt.
Brighton	Nicholson, John V.	Capt.
Brockton	Mason, Carl	Capt.
Cambridge	Yaffee, Joseph J.	Capt.
Charlestown	Gordon, David S.	Capt.
Chelsea	Rosenfield, Jack L.	Capt.
Dorchester	Luresky, Samuel S.	Capt.
Dorchester	Meiserlman, Harry	Capt.
East Lynn	Emery, Norman A.	Capt.
Fitchburg	Winthrop, Harry	Capt.
Greenfield	Grayson, Maurice B.	Capt.
Hingham	Vroom, Ross, Jr.	Capt.
Holyoke	La Belle, W. M., Jr.	Maj.
Holyoke	Marran, V. P., Jr.	Maj.
Hyannis	Kepnew, Benjamin	Capt.
Jamaica Plain	Leathe, Wilbor, Jr.	Capt.
Lynn	Murray, James I.	Capt.
Milford	Helfand, Harry	Lieut.
Nahant	Nichols, John H.	Lieut.
Needham Heights	Pope, Richard O.	Capt.
Newton Center	Grossman, Israel W.	Capt.
Pittsfield	Levy, Arthur M.	Capt.
Pittsfield	Shelsy, Michael J.	Capt.
Roxbury	Feldman, Irving	Capt.
Roxbury	Goldsmith, William E.	Capt.
Roxbury	Gordon, Maxwell N.	Lieut.
Roxbury	Hoffman, William M.	Capt.
Roxbury	Jacobs, Harvard D.	Capt.
Roxbury	Yavner, Max	Capt.
Springfield	Little, Thomas G.	Maj.
Weymouth	Danehy, Alfred F.	Capt.
Whitinsville	Horan, James F.	Capt.
Worcester	Higgins, Lemuel W.	Maj.

Michigan

Bangor	Campbell, Joseph M.	Capt.
Battle Creek	Valluzzo, Rocco C.	Maj.
Bay City	McManmon, E. P.	Maj.
Columbiaville	Fellows, Birney F.	Capt.
Dearborn	Westmoreland, W. W.	Capt.
Detroit	Glynn, Martin A.	Capt.
Detroit	Katz, Ben J.	Lieut.
Detroit	Kellog, Richard G.	Capt.
Detroit	Kowal, Michael G.	Lieut.
Detroit	Sherman, Phillip M.	Capt.
Grand Rapids	Glerum, Simon, Jr.	Capt.
Hillsdale	Anthony, Kenneth W.	Capt.
Ironwood	Velin, Verner H.	Capt.
Kalamazoo	Hawson, Thomas W.	Capt.
Lansing	Dillingham, S. M.	Capt.
Marine City	Dunklin, Volney L.	Maj.
Mt. Pleasant	Woodruff, Howard R.	Capt.
Nazel Park	Hewitt, Robert D.	Capt.

Minnesota

Atwater	Gilman, Donald O.	Capt.
Chisholm	Banen, Abraham T.	Capt.
Duluth	Peterson, Ray L.	Capt.
Duluth	Tornstrom, C. H.	Capt.
Keewatin	Loofbourrow, R. H.	Capt.
Minneapolis	Beck, Rolland C.	Capt.
Minneapolis	Brecht, Lyle A.	Maj.
Minneapolis	Gimpel, Samuel G.	Capt.
Minneapolis	Johnson, Carl L.	Capt.
Minneapolis	Lawson, Ansel M.	Capt.
Minneapolis	Nelson, Earle W.	Lt. Col.
Minneapolis	Paulsen, John M.	Capt.
Minneapolis	Svendsen, Gustav	Capt.
Owatonna	Liedl, Eugene J.	Capt.
Raymond	Kleinhuizen, C. M.	Maj.
Red Wing	Edgerton, Karl A.	Maj.
Rochester	Miner, Robert E.	Capt.
Rochester	Sund, Frederick R.	Lt. Col.
St. Paul	Hopkins, John L.	Capt.
St. Paul	Hovland, Stanley J.	Capt.
St. Paul	MacCraw, Robert M.	Maj.
St. Paul	Milstein, Norman	Capt.
St. Paul	Nelson, George K.	Capt.
St. Paul	Pearlman, Joseph	Capt.
St. Paul	Windorfer, John H.	Lieut.
Sauk Centre	Johnson, Lyle T.	Capt.
Slayton	Daley, Eugene P.	Capt.
Wabasha	Schroeder, Tylas R.	Capt.
Windom	Wilson, Harold A.	Lt. Col.
Zumbrota	Woodbury, Wayne L.	Capt.

Mississippi

Columbus	Broadfoot, John C.	Capt.
Jackson	Cohen, Bernard A.	Capt.
Jackson	Russ, Curtis R.	Capt.
Jackson	Weaver, Charles A.	Maj.
Meridian	Walker, Oscar J.	Maj.
Water Valley	Patton, Coy E.	Capt.

Missouri

Cole Camp	Taylor, Lewis A., Jr.	Capt.
Fulton	Crews, Carrol K.	Lt. Col.
Kansas City	Barger, Lloyd M.	Lt. Col.
Kansas City	Browning, Anthony	Capt.
Kansas City	Gossett, John E.	Capt.
Kansas City	Gottlieb, Meyer P.	Maj.
Kansas City	Kushner, Abe L.	Capt.
Kansas City	Siegelbaum, William	Capt.
Montgomery City	Ball, Richard B.	Maj.
Ozark	Shallenberger, J. N.	Capt.
St. Louis	Anscombe, Harry L.	Lieut.
St. Louis	Hamilton, Merritt C.	Capt.
St. Louis	Hessee, Walter A.	Capt.
St. Louis	Kloetzer, Adelbert O.	Capt.
St. Louis	Linders, Harold Z.	Capt.
St. Louis	O'Brien, Lane W.	Maj.
St. Louis	Shepard, Earl E.	Lt. Col.
St. Louis	Wagman, Leonard	Capt.
St. Louis	Walker, Donald J.	Capt.
Savannah	Brown, Eugene I.	Maj.
Webster Groves	Robinson, Laurance H.	Maj.
Wellsville	McMurtry, Don C.	Capt.

Montana

Bigtimber	Olness, Bernhard	Capt.
Billings	Shaver, Deane S.	Capt.
Butte	O'Brien, Murray J.	Capt.
Cut Bank	Yunck, Harry R.	Capt.
Fort Harrison	Jones, Guy H.	Capt.

Nebraska

Auburn	Harding, Albert T.	Capt.
Omaha	Free, Jack C.	Lieut.
Omaha	Hornbeck, D. L.	Lt. Col.
Omaha	Molak, Frank R.	Capt.
Omaha	Randall, William F.	Capt.
Pierce	Rysavry, Lumer J.	Maj.

New Jersey

Atlantic City	Roche, Saul	Capt.
Belleville	Parrillo, Anthony C.	Capt.
Bloomfield	Ferrairs, Joseph F.	Capt.
Bloomfield	Fischer, Meyer	Capt.
Carteret	Greenwald, Leon	Capt.
Collingswood	Donohue, Thomas V.	Capt.
Englewood	De Scherer, Morton	Capt.
Gloucester	Brown, William	Maj.
Jersey City	Kurtz, Milton M.	Capt.
Montclair	Finkel, David	Capt.
Newark	Gray, Noah	Capt.
Newark	Kaminsky, Ira I.	Maj.
Newark	Neger, Milton	Capt.
Orange	Engel, Walter	Capt.
Orange	Pachias, Philip H.	Capt.
Paterson	Spector, Norman B.	Capt.
Trenton	Hatrak, Nicholas E.	Capt.
Wrightstown	Weil, Carlos	Maj.

New York

Albany	Gorski, Alexander F.	Maj.
Brooklyn	Blaustein, Samuel	Capt.
Brooklyn	Cervavolo, Joseph J.	Capt.
Brooklyn	Geller, George B.	Lt. Col.
Brooklyn	Goodman, Sidney W.	Capt.
Buffalo	Grovino, Joseph D.	Capt.
Cheektowaga	Ciesiel, Edmund K.	Lieut.
Flushing	Blumenthal, A. B.	Lieut.
Glens Falls	Hughes, Harry J.	Capt.
Hawthorne	Gluskin, Harry	Lieut.
Hempstead	De Clue, Bertrand L.	Maj.
Long Beach	Cohen, Arthur	Capt.
Mt. Vernon	Ash, Arthur S.	Capt.
New York	Andrioli, Albert N.	Lieut.
New York	Bailin, Jacob B.	Capt.
New York	Brown, Monroe S.	Capt.
New York	De Vanna, Alonza N.	Capt.
New York	Goldman, George C.	Capt.
Schenectady	Bucci, John T.	Lieut.
Troy	Hunn, David E.	Maj.

North Carolina

Chandler	Rich, Charles F.	Capt.

Ohio

Canton	Hupp, Lloyd H.	Lt. Col.
Cleveland	Foote, James E.	Capt.
Cleveland	Gilden, Bernard B.	Maj.
Cleveland	Lavergna, Michael P.	Lieut.
Columbus	Berger, Norman B.	Capt.
Columbus	Blasberg, Everett G.	Capt.
Columbus	Cabakoff, Izador I.	Capt.
Columbus	Denlinger, Arthur J.	Capt.
Columbus	Franklin, W. W.	Lt. Col
Columbus	McCarron, Robert F.	Maj.
Dayton	Armacost, James H.	Maj.
Kenton	Jerles, Robert F.	Maj.
Lakewood	Cichowicz, Stanley A.	Capt.
Lima	Fly, Charles A., Jr.	Capt.
Lima	Miller, Roger W.	Capt.
Minerva	Bates, Ralph	Capt.
Newark	Kennedy, George T.	Lieut.
Portsmouth	Barry, Ray J.	Capt.
Youngstown	Frank, Albert E.	Lieut.
Youngstown	Malkoff, Abe	Capt.

Oklahoma

Hollis	Bell, L. H.	Maj.
Hominy	Blum, Charles J.	Capt.
Hooker	Bocock, Alman E.	Maj.
Muskogee	Overmyer, Glenn W.	Capt.
Okmulgee	Grove, Arthur W.	Capt.
Tulsa	Moore, Allen H.	Capt.

Oregon

Eugene	Carson, Raefield A.	Capt.
Portland	Jeffcott, Robert L.	Maj.
Portland	Wollin, Sanford C.	Maj.

Pennsylvania

Custer City	Dixon, Walter R.	Maj.
Lansdowne	Owen, Edwin G.	Capt.
Lowanda	MacLaren, Robert J.	Capt.
Mahoningtown	Mrozek, Francis P.	Capt.
New Castle	Jackson, William F.	Lt. Col.
Perrysville	Atkinson, David F.	Capt.

Philadelphia	Dienstein, Benjamin	Capt.
Philadelphia	Field, Benjamin J.	Capt.
Philadelphia	Gimbel, A. B., Jr.	Lieut.
Philadelphia	Schupack, Isador B.	Maj.
Philadelphia	Silverman, Louis J.	Capt.
Philadelphia	Stevens, Ford W.	Maj.
Philadelphia	Yanoff, Harry L.	Capt.
Pittsburgh	Hauman, Adam J.	Capt.
Pittsburgh	Schugar, George	Lieut.
Washington	Hoop, Elmer P.	Capt.

South Dakota

Britton	Aistrup, Carl G.	Capt.
Hot Springs	Croup, Oren F.	Capt.
Lennox	Straup, Perry D.	Capt.
Tripp	Misterek, Emil A.	Capt.

Tennessee

Knoxville	Book, William H.	Capt.
Nashville	Bergman, Sylvester H.	Capt.
Union City	Gibbs, Homer E.	Maj.

Texas

Arlington	McMinn, Jacques S.	Maj.
Austin	Carrington, Joe C., Jr.	Capt.
Beaumont	Walker, Howell H.	Maj.
Corpus Christi	Neehan, Thomas C.	Capt.
Dallas	Goodman, Thomas K.	Capt.
Dallas	Nellen, Herman A.	Capt.
Dallas	Young, Amo C.	Capt.
Fort Worth	Drechsel, R. H. W.	Lt. Col.
Galveston	Del Papa, Torello A.	Capt.
Kenedy	Avery, Roy H.	Capt.
Marshall	Munden, Willie T.	Capt.
Mt. Vernon	Stenson, David L.	Capt.
San Antonio	Harvey, George G.	Maj.
San Antonio	Maurer, Leslie D.	Lt. Col.
San Antonio	O'Quinn, Edgar	Maj.
Stamford	Rutherford, Joseph H.	Maj.

Utah

Midvale	Rigby, Milton T.	Capt.
Ogden	Pincock, Douglas F.	Maj.
Salt Lake City	Call, Everard C.	Maj.
Salt Lake City	Sampson, Bernard L.	Maj.

Virginia

Bakerville	Young, Robert B.	Capt.
Norfolk	Mendelsohn, Harry B.	Capt.
Portsmouth	Bayton, E. L., Jr.	Capt.
Richmond	Sittler, Werner G.	Capt.
Scottsville	Jone, James A.	Capt.

Washington

Bellingham	Syre, Ray S.	Capt.
Hoquiam	Meyer, Albert H.	Capt.
Seattle	Deines, John M.	Maj.
Seattle	McClain, John H.	Capt.
Seattle	Smith, Francis F.	Maj.
Yakima	Severn, Charles A.	Capt.
Yakima	Sims, Francis A.	Capt.

West Virginia

Fairmont	Laughlin, Carl A.	Maj.
Monongah	Shenasky, John H.	Capt.
Morgantown	Cunningham, C. E., Jr.	Capt.
Parkersburg	Freed, Donald G.	Capt.
West Huntington	Timken, William H.	Capt.

Wisconsin

Appleton	Chudacoff, Sidney L.	Capt.
Campbellsport	Romaine, Frank E.	Capt.
Gilman	Slota, Bernard R.	Capt.
Green Bay	Kelly, Norman P.	Capt.
Green Bay	Trace, Lester	Capt.
Madison	Reinke, Gilmer A.	Capt.
Madison	Sannes, Dedrick	Capt.
Manitowoc	Toepel, Robert G.	Lieut.
Milwaukee	Fogelson, Samuel S.	Capt.
Milwaukee	Gehardt, George C.	Capt.
Milwaukee	Knudten, John R.	Capt.
Milwaukee	Schwager, Rubin D.	Capt.
Milwaukee	Van Dale, Dwight W.	Lieut.
Oshkosh	Hinz, Otto B.	Lt. Col.
Plymouth	Licking, Ralph A.	Maj.
Whitefish Bay	Halberstadt, Henry J.	Capt.

Wyoming

Casper	Ebinger, Frederick W.	Capt.
Casper	Hocker, William A.	Capt.
Hanna	Hoffman, Arthur J.	Capt
Sheridan	Bailey, George W.	Capt.

BOOK REVIEWS

Man Against Pain: The Epic of Anesthesia. By Howard Riley Raper, D.D.S. 337 pages. Illustrated. Bibliographical Appendix. Index. Cloth. Price $3.50. New York: Prentice-Hall, Inc., 1945.

The tragic, fascinating and often sordid story surrounding the discovery of anesthesia has been told so often by advocates, scenario writers, historians, partisans and fiction writers that one might well question the value of a reconsideration of the subject. Too often in the past, writers on this topic have been carried away from an objective exposition by the need to espouse a cause or defend a reputation. "Man Against Pain" has escaped these twin pitfalls because the author rigidly marshals the best available evidence toward sound conclusions. This is a book, therefore, that has the dramatic pace of a detective story and the critical attitude of truly scientific writing. There is some new material, but the real value of the book lies in the author's evaluation of the evidence.

On the celebrated question of priority for Horace Wells or W. T. G. Morton, Raper says:

> One reason why it has been so difficult to name the discoverer of anesthesia is that *there is no discoverer of anesthesia!* No one man earned exclusive right to the title. The *discoverers* are Wells *and* Morton—Wells, in the sense that he found it and passed it along to Morton; Morton, in the sense that he introduced it into surgical practice. Wells received the ball from center and passed it to Morton who stepped over for a touchdown. It is highly improbable that there would have been much controversy if Morton had acknowledged his indebtedness, for no other candidate could have won any significant recognition against such a combination based securely on truth. (Italics in the original.)

"Man Against Pain," which has been a selection of the Scientific Book Club, does not limit itself to the controversy but tells the entire story of man's conquest of pain by hypnotism, narcotics and local and general anesthetics. There is also a section devoted to some of the newer anesthetics whose wider use was given impetus during the war. The short sections on dreams could have been eliminated with no loss either in effectiveness or in completeness. More than forty pages of critical bibliography, set apart in an appendix so as not to interfere with the progress of the story, comprise a valuable contribution to the involved history of the subject.

The author tells his story dramatically and convincingly. The style is sprightly and fast-paced enough to interest the lay reader as well as the dentist and physician. Occasionally, the organization of the material seems sketchy, and the illustrations certainly do not match the high quality of the text. These are minor defects, however, that do not impair the book's very great value as intelligent entertainment and as a solid contribution to the history of anesthesia. Raper, himself a dentist, has finally set Horace Wells and the others involved in the controversy in proper perspective. Because so many other authors have failed in this job, Raper's book can be considered as an achievement of the highest order.

School Health Policies. By the National Conference for Cooperation in Health Education. 46 pages. Second Edition. Price $0.25. New York 14: Health Education Council, 10 Downing St., 1945.

This excellent booklet should be in the hands of all persons interested in the development of health activities for children and adults. The recommendations for school health policies were for-

mulated by specialists in the health field representing school systems and the dental, medical, nursing and public health professions.

In far too many instances, recommended procedures for the promotion of health and physical education are indefinite and impractical and are not based on a comprehensive knowledge of the problems involved. This booklet, on the other hand, presents clearly stated and practical suggestions for improved school health programs. While the suggested procedures are directed to the school administrator, they are also addressed to all persons concerned with the health of the school child, so that these persons too may cooperate in carrying out programs for the health of students and, indirectly, of all members of the community.—*Allen O. Gruebbel.*

The Health of a Nation. An analysis by Michael M. Davis, Chairman, Committee on Research in Medical Economics, New York City, and Bernhard J. Stern, Department of Sociology, Columbia University. Teaching Aids by Lavone A. Hanna, Acting Assistant Professor of Education, Stanford University. 84 pages. Price $0.30. Washington 6, D. C.: National Education Association, 1201 Sixteenth St., N.W.

"The Health of a Nation" is number 17 of a series of pamphlets entitled "Problems in American Life," prepared under the joint sponsorship of the National Council for the Social Studies and the National Association of Secondary School Principals, which are departments of the National Education Association.

Although "The Health of a Nation" is prepared primarily for teachers in secondary schools, dentists and others interested in the social and economic aspects of the nation's health problem will gain much by reading it. Part one consists of an analysis by Michael M. Davis and Bernhard J. Stern. Its seven short chapters present information on the nature of the problem; the individual's health; health and the community; health technology and economics; processes and goals; reading references, and agencies providing information. The authors point out that individuals have the important responsibility of looking after their own health, but that health is also a concern of society. "In what degree, by what methods, through what agencies," they ask, "should control be exercised by group action over the elements that affect health and over the institutions and vocations that deal with it?" Under the general subject of individual health, mental health, foods, food fallacies, physical exercise, rest, cleanliness, ventilation and avoidance of harmful drugs are discussed. The chapter on community health presents some vital information on communicable disease, changing birth and death rates, life expectancy, health and income, industrial accidents and occupational diseases. Information on health service personnel and their geographic distribution is presented. The number and distribution of hospitals are discussed. The nation's health bill, supply of and demand for health services, physicians' and dentists' incomes, private and public health agencies and health legislative proposals are presented in a clear, understandable manner.

Dentists interested in socio-economics will like Part one of this booklet, although they will regret that it does not contain more information on the dental aspect of the problem.

The only serious criticism that might be directed at "The Health of a Nation" is its lack of dental statistics. Two serious typographical errors were noted.—*Lon W. Morrey.*

CURRENT LITERATURE

Mixed Tumors of the Salivary Glands. C. A. Hellwig. Arch. Path. 40:1, July 1945.

In eighty-four primary tumors of the salivary glands occurring among 4,352 cases of cancer over a period of twenty years, the average age of patients with mixed tumors was 38.4 years. More women were operated on for mixed tumor and more men for true cancer of the salivary glands. Sixty-eight (82 per cent) tumors were in the parotid glands, twelve in the submaxillary glands and three in the palate. None were found in the sublingual glands. Histologically, fifty-one of the eighty-two tumors were of the mixed type and sixteen of the adenomatous type. Eight presented combinations of these two types and seven were true cancers.

The salient feature of mixed tumors is the transformation of epithelial cells into myxochondroid tissue. The origin of tumor cells, which can appear to be epithelial and again mesenchymal, is not satisfactorily explained by the three current theories of histogenesis of mixed tumors of the salivary glands. From embryologic, histologic and topographic studies, it was concluded that these tumors are derived from misplaced elements of the notochord (chorda dorsalis). This theory would explain not only the complex structure of mixed tumors, but also the striking predilection of these tumors for the parotid glands.

Preparation of a Penicillin Lozenge. J. A. Nussle. Am. J. Pharmacol. 117:272, August 1945.

A lozenge base suitable for the preparation of a penicillin lozenge must (1) slowly dissolve in the saliva when held between the tongue and the hard palate; (2) remain sufficiently fluid to pour at temperatures between 60 and 70 C.; (3) have a pleasant taste and flavor, and (4) contain no ingredient which will cause deterioration of the penicillin. Two lozenge bases meeting these requirements have been developed. The first is a glycerinated gelatin base; the second is a gel prepared from agar, simple syrup and glycerite of starch. The former dissolves in ten minutes and the latter in twenty minutes. Both lozenges retain satisfactory antibacterial activity for two to three weeks when refrigerated at 5 C.—*J. E. Gilda.*

Field Study of the Prevalence of the Clinical Manifestations of Dietary Inadequacy. W. J. Darby and D. F. Milam. Am. J. Pub. Health 35:1014, October 1945.

Clinical findings in a nutritional survey of 959 individuals representative of the rural population of Alamance County, N. C., covered physical examinations, medical history, laboratory procedures and seven-day food intake records. Especial attention was given to corneal vascularization, angular fissures of the mouth, tongue changes, gingivitis, neurologic alterations, etc. Of twenty-six persons with angular fissures, only one had a recorded intake of less than 0.7 mg. of riboflavin. The others showed adentia, wore ill-fitting dentures, etc. Tongue changes simulating ariboflavinosis were found in three adults, two of whom had anemia and the other a riboflavin intake of 1.1-3.3 mg. per day. Asymptomatic changes in the buccal mucosa, found in 16 per cent of adults and 2.2 per cent of children, could not be correlated with riboflavin deficiency. No cases of the scarlet red tongue of niacin

deficiency were seen. Marginal gingivitis, seen in 309 subjects, seemed to be related to poor oral hygiene and not to vitamin C deficiency. The most prevalent definite abnormalities attributed to malnutrition were underweight, obesity and low hemoglobin index. The evidence presented does not support the thesis that any serious dietary deficiency existed in this population.—*Paul H. Keyes.*

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Titles marked with an asterisk are reviewed below.

*Oral Focal Infection: Its Status Today, with Particular Reference to Arthritis. Dwight C. Ensign, M.D., Detroit, Mich.—89.

*Periodontal Cysts. Edward C. Stafne, D.D.S., and Jerry A. Millhon, D.D.S., Rochester, Minn.—102.

*Early and Late Treatment of Gunshot Wounds of the Jaws in French Battle Casualties in North Africa and Italy. John Marquis Converse, M.D., New York, N. Y.—112.

*Combined Pentothal Sodium-Nitrous Oxide-Oxygen Anesthesia in Oral Surgery. Orlan K. Bullard, D.D.S., and Sheldon I. Brockett, D.D.S., San Diego, Calif.—138.

*Control of Submandibular Cellulitis by Penicillin Therapy: Report of Case. Leon J. Allen, D.D.S., Mount Vernon, N. Y.—152.

*New Technic for Correction of Macrostomia. Harold I. Harris, M.D., Hollywood, Calif.—156.

*Stenosis of the Oropharynx. Varaztad H. Kazanjian, D.M.D., M.D., Boston, Mass.—164.

*Myositis Ossificans of Right Masseter Muscle: Report of a Case. James R. Cameron, D.D.S., and J. J. Stetzer, Jr., D.D.S., Philadelphia, Pa.—170.

*Temporary External Salivary Drainage (from Stenson's Duct) Following Arthroplasty. J. Orton Goodsell, D.D.Sc., Saginaw, Mich.—174.

Oral Focal Infection: Its Status Today, with Particular Reference to Arthritis. Dwight C. Ensign.

It is Ensign's intention to clarify the relation of oral focal infection to arthritis. He says: "Much of the apparent confusion and muddled thinking on the subject of focal infection results have their bases in several facts: First, that there is no clear-cut proof of the mode of action of a focus of chronic infection in causing disease at a distance. In some instances, metastasis of bacteria seems to occur; in others, the absorption of toxic products is thought to be important; while, in others, the existence of a state of allergy is postulated. A second fact is that many, perhaps most, of the conditions in which focal infection has been thought to play a part are diseases the exact etiology of which is still uncertain or unknown, and, as we shall indicate, focal infection is but one of many factors in the particular problem. Thirdly, some still fail to differentiate between cause and effect, between early changes and organized tissue changes; and the failure of established disease to be modified by eradication of a focus is unjustly held to be evidence against the original influence of the focus in initiating such changes. A fourth source of confusion consists in the making of general statements, rather than in confining observations to one disease or to one group of diseases." Therefore, diseases other than arthritis are not discussed. Patients who make self-diagnoses of arthritis should be adequately examined to determine whether the disease is arthritis and, if so, which type is present. If it is nonspecific, is it chronic infectious arthritis or hypertrophic arthritis? In both types, the following etiologic categories are considered: (1) heredity; (2) nutrition; (3) glandular and metabolic conditions; (4) mechanical states (weight, etc.); (5) personality, and (6) infectious conditions (including all possible foci). Hypertrophic joint disease is more common than infectious arthritis. It is largely a matter of "wear and tear." The treatment of oral infection in this type should be the same as though no joint disease were present. We should not be radical. While no definite proof exists that focal infection is responsible for chronic infectious or rheumatoid arthritis, we must suspect such a relationship and be more radical in our elimination of possible foci. Residual roots with radiographic translucency are frequently overlooked. Rare indeed is the arthritis which can be cleared up solely by elimination of a focus. A group or team, one being a dentist, serves best in diagnosis

and treatment. Infection should be eliminated from "normal" people. X-ray films of tooth-bearing and edentulous areas should be made. Root canal surgery, from the medical point of view, is permissible if no systemic disease is present. Nonvital teeth should be removed if a progressive systemic disease that does not respond to other treatment is present. Periodontoclasia is important, particularly when extended to noneradicable crypts at root bifurcations. Physician-dentist teamwork is essential. Generally speaking, the activity of the disease should be under control before surgery is instituted. The amount of surgical work to be performed at any one time is a matter for team decision. Preextraction sulfonamides are useful in reducing bacteremia, which may be harmful to rheumatic hearts. Penicillin may prove to be of greater benefit than the sulfonamides in prophylaxis against endocarditis.

Periodontal Cysts. Edward C. Stafne and Jerry A. Millhon.

Stafne and Millhon classify periodontal cysts as root-end, lateral or residual types. A cyst may develop from residual infection not demonstrable at the time of extraction. The incisor-cuspid region produces many more cysts than other areas, the maxillary lateral incisor accounting for more than one-third of those tabulated. The possibility of cyst development on untreated pulpless teeth is greater than if the root-canal is treated. "Roentgenograms of thirty-six cysts were made at intervals ranging from fourteen months to fifteen years after first observed. In fifteen of the thirty-six instances, it was noted that the cyst had increased in size; in seventeen instances, there was no change in size or roentgenographic appearance; in three instances, the cyst had decreased in size, and, in one instance, it had disappeared completely. In some instances, a cyst does not reach its maximal size until the cortical plate has been perforated and drainage to the external surface has been established. In the majority of cases, growth is limited, and once there is a cessation of growth, probably only rarely is there further increase." Rarely do cysts disappear spontaneously. When they do, the condition responsible is, undoubtedly, drainage, although the avenue may not be detectable.

Early and Late Treatment of Gunshot Wounds of the Jaws in French Battle Casualties in North Africa and Italy. John Marquis Converse.

Converse reports his treatment of gunshot wounds of the jaws while he was associated with the French in North Africa and Italy. Early and late care are described and illustrated. "The treatment of gunshot wounds of the jaws may be divided into three distinct periods. The first, or early, phase, which extends from the time of injury to one or two days after injury, is the most critical for the patient. This is the period of asphyxia, loss of blood, shock, pain and mental depression. After this critical first period, the patient enters the second phase, during which, in accordance with the severity of the case, the treatment of the patient consists in the immobilization of the bone fragments by means of ligatures or splints, to increase his comfort and to control infection, which may result in pulmonary complications or secondary hemorrhage. The late primary suture of the wounds may be carried out in this prereconstructive phase. The third period is the reconstructive phase proper, which follows healing of the tissues and often evacuation of the patient to the zone of the interior. At this time, reconstructive plastic operations, often interrupted by periods of convalescence, are started." Hemorrhage and shock control plus relief of respiratory destruction constitute treatment in the first critical period. Further treatment is administered by the maxillofacial team at the next station when possible. Primary suture is inadvisable. "While débridement of facial wounds should be sparing, surgical cleansing should strive to remove small devitalized tags of tissue, loose and detached bone fragments lying in the wound and foreign bodies. Detached flaps, hanging by gravity, should be supported by dressings held by bandages. The most important point in the early treatment of gunshot wounds is to provide immobilization of the remaining fragments by simple means, such as ligature wires or elastics. Reduction of the fragments is easy at this stage. The restoration

of the normal anatomic position of the soft tissues is aided when displaced fragments of bone are restored to their normal position of dental occlusion. Large gaping wounds into which displaced fragments are projecting become smaller and restoration of the normal position of the remaining structures facilitates future reconstruction work." Next, feeding problems are conquered and definitive treatment is begun, such as bone and soft tissue immobilization, control of infection with chemotherapy, removal of débris, etc. Within seven to fourteen days, late primary suture may be carried out, but only after bone fragments have been thoroughly immobilized. Acrylic splints or molds are placed as foundations for soft tissue repair if bone loss is large. Skin edges are sewed to mucous membrane edges when necessary. Discretion must be used in the extraction of teeth which are in lines of fracture but, generally speaking, such teeth should be removed. External drainage is employed for relief of pus or hematomatous accumulations only. External fixation was used rarely and then largely on edentulous areas. The reconstructive phase is greatly simplified by proper treatment in the first two stages.

Combined Pentothal Sodium-Nitrous Oxide-Oxygen Anesthesia in Oral Surgery. Orlan K. Bullard and Sheldon I. Brockett.

Bullard and Brockett combine pentothal-sodium and nitrous oxide-oxygen to advantage in anesthesia for oral surgery. The barbiturate is used intravenously for induction and the operation is carried out from there on with nitrous-oxide 75 per cent and oxygen 25 per cent. Decompensated cardiac disease, dyspnea, limitation of respiratory motion, etc., are considered contraindications. Shallow anesthesia and deep infections of the neck may also be deterrents. If atropine is used preoperatively and oxygen is employed in sufficient amounts, with slow and adequate induction, metrazol or atropine is available, depth of anesthesia is maintained, tracheotomy equipment is handy, etc., Ludwig's angina may be considered a proper field for employment of pentothal and nitrous-oxide. Children seemed to do well. Morphine premedication should be avoided in the young or frail. Elixir pentobarbital sodium, one-fourth grain per dram, is fine in children. A 3-year-old child receives 3 drams with one one-hundred-fiftieth grain of atropine. Hypotensive patients receive neosynephrine, 0.5 cc. of a 1 per cent solution, intramuscularly to raise the pressure. From 20 to 30 cc. of 2.5 per cent pentothal is all that normally is used. After induction, a nasal inhaler is applied and the nitrous oxide-oxygen is administered. Additional pentothal is given as needed during operation.

Control of Submandibular Cellulitis by Penicillin Therapy: Report of Case. Leon J. Allen.

Allen reports a case of submandibular cellulitis which responded dramatically to penicillin therapy after treatment with the sulfonamides had not been successful.

New Technic for Correction of Macrostomia. Harold I. Harris.

Harris' repair of macrostomia uses a new technic, which can be understood easily if the diagrams and illustrations are seen. It involves the use of vermilion flaps at the angle of the mouth and proposes to eliminate postoperative scarring and fissuring, which are common after other corrective procedures.

Stenosis of the Oropharynx. Varaztad H. Kazanjian.

The usual cause of extreme narrowing of the oropharynx can be found in some postoperative tonsillectomies. Kazanjian cautions against loss of epithelium between the faucial and lingual tonsils. The correction is made by freeing the pillar and covering the denuded surface with flaps from the nearby retromolar area and cheek.

Myositis Ossificans of Right Masseter Muscle: Report of a Case. James R. Cameron and J. J. Stetzer.

Cameron and Stetzer report myositis ossificans of the masseter muscle, the only such case which the writers believe has been reported. The patient, a colored man, aged 28, fell and struck the side of his face. Two months later, trismus and swelling were present. A bony mass, 2.5 cm. in length,

was removed. Function and comfort returned. One month later, there was a recurrence of signs and symptoms. Six months later, after the process had been allowed to go on to completion, the whole masseter, bonelike, was chiseled from the mandible and zygoma and removed. Function returned. Six months later, more calcified fragments were removed from the area. The patient has not been seen since then.

Temporary External Salivary Drainage (from Stenson's Duct) Following Arthroplasty. J. Orton Goodsell.

In the case reported, the duct was apparently encountered either by a Gigli saw or by the opening on a Dingman type pedicle needle. Tight adhesive tape over the opening on the cheek caused the saliva to return to its normal course via the terminal portion of the duct.

Cancrum Oris. J. Dawson. Brit. D. J. 79:11, September 1945.

From observations and treatment of ten cases of cancrum oris seen among internees of the Belsen concentration camp, the author concludes that this disease, originating from an ulcerative stomatitis, which spreads to involve the lips or cheeks, occurs as a complication of extreme debilitation. Local penicillin therapy will either prevent, or arrest the spread of, the disease and will allow normal healing if the general condition of the patient permits recovery.—*Paul H. Keyes.*

Vincent's Disease of the Skin. A. Stricker. Arch. Derm. & Syph. 52:87, August 1945.

In a case of Vincent's disease of the skin involving the feet and the oral cavity, Vincent's organisms, although demonstrated in oral lesions, were not observed in the toenails or paronychial tissue. Successful treatment consisted of application of sodium perborate to the feet and neo-arsphenamine to the gums. Thiobismol was given intramuscularly. A positive Wasserman reaction is regarded as incidental and not the result of the Vincent's infection.—*Paul H. Keyes.*

Obturator (Acrylic) for the Newborn Infant with Cleft Palate. I. E. Shapiro, A. Walden and G. A. Carman. J. Pediat. 27:288, September 1945.

In two cases of new-born infants with cleft palates, feeding was made possible by the use of acrylic obturators. A low-melting modeling compound was used to take the impressions. The obturators were well tolerated and no difficulty in inserting or removing them was reported. —*Paul H. Keyes.*

Erythema Multiforme. H. C. Robinson. Arch. Derm. & Syph. 52:91, August 1945.

In a severe case of erythema multiforme in a 15-year-old boy, there was almost complete involvement of the buccal mucosa, tongue and lips. The gingiva and pharyngeal wall were red and swollen, but not covered with vesicular lesions. Continuous diarrhea and vomiting caused severe complications. On a purely empiric basis, penicillin was given intramuscularly for four days, a total of 760,000 units. The dramatic drop in temperature and the rapid recovery after the initiation of penicillin therapy seemed sufficiently significant to warrant reporting the observation.—*Paul H. Keyes.*

Two Types of Undermining Dentin Caries. Bernhard Gottlieb. J. Am. Coll. Dent. 12:73, June 1945.

Of two forms of caries producing undermining at the dentinoenamel junction, one presents a spread of yellow pigmentation beneath intact enamel, apparently the result of entrance of proteolytic micro-organisms into the dentin by way of the lamellae. The other type shows dentin shrinkage, presumably the result of invasion of prism sheaths by acid-producing micro-organisms.—*Paul H. Keyes.*

DEATHS

Abbott, Chester R., Des Moines, Iowa; State University of Iowa, College of Dentistry; died November 17; aged 50.

Ames, V. B., Baltimore, Md.; died May 11.

Anderson, Harold E., Hoboken, N. J.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania; died November 25; aged 53.

Anderson, Johan, Miami, Fla.; died October 29; aged 51.

Argall, Frank L., San Jose, Calif.; died September 1.

Bachman, Harry P., Decatur, Ill.; Pennsylvania College of Dental Surgery, 1895; died October 4; aged 72.

Baker, George C., Elkins, W. Va.; Baltimore College of Dental Surgery; died October 20; aged 62.

Balderston, E. B., Springfield, Mass.; died in 1945.

Beckford, Fred A., Boston, Mass.; Harvard University Dental School, 1906; died November 7.

Bell, George E., Fergus Falls, Minn.; University of Chicago Dental College, 1904; died November 12; aged 55.

Berens, Val H., Glencoe, Minn.; University of Minnesota, School of Dentistry; died November 18; aged 54.

Besore, C. F., Ida Grove, Iowa; died November 1; aged 74.

Bierke, Edward C., Denmark, Wis.; Marquette University Dental School; died September 6; aged 44.

Billig, Hal, Maywood, Ill.; died November 7; aged 76.

Blaisdell, Edwin Carter, Portsmouth, N. H.; Harvard University Dental School, 1883; died November 19; aged 84.

Block, W. B., Meridian, Miss.; died October 30.

Bretz, Arthur H., San Diego, Calif.; died October 28; aged 76.

Bridgman, E. P., Madison, Wis.; Northwestern University Dental School, 1903; died November 13; aged 65.

Bronson, David J., Bronx, N. Y.; died November 19; aged 60.

Brown, P. H., El Paso, Texas; Northwestern University Dental School; died November 30; aged 74.

Buck, W. H., Orangeville, Ill.; died November 5; aged 45.

Butler, Gerald L., Lincoln, Nebr.; died December 1; aged 47.

Buttner, Henry F., Coeur d'Alenes, Idaho; Baltimore College of Dental Surgery; died September 18; aged 87.

Campbell, John Murdock, Mobile, Ala.; died in October; aged 83.

Canine, Robert F., Louisville, Ky.; University of Louisville, School of Dentistry; died in 1945; aged 67.

Carlaw, A. C., Northfield, Minn.; died in 1945.

Carmichael, Joseph S., Cambridge, Mass.; Tufts College Dental School, 1895; died October 10; aged 70.

Casey, James F., Clearwater, Fla.; died October 23; aged 70.

Cassell, Leonard C., Decatur, Ill.; died November 5.

Claxon, William O., Lexington, Ky.; University of Louisville, School of Dentistry; died November 7; aged 53.

Cleary, George C., Elizabeth, N. J.; Tufts College Dental School; died December 5; aged 52.

Cottrell, Arthur J., Knoxville, Tenn.; Tazewell College, O-T Dental Department; died October 31; aged 84.

Cowan, James H., Cortland, N. Y.; Baltimore College of Dental Surgery; died November 4; aged 76.

Crowe, Hilliard, Detroit, Mich.; died November 16; aged 70.

Culbertson, Howard, Germantown, Pa.; died December 4.

Dillon, William H., Zanesville, Ohio; died December 8; aged 70.

Doolittle, Lewis T., Milford, Conn.; Baltimore College of Dental Surgery; died in October.

Douglas, Arthur H., Indianapolis, Ind.; died October 25.

Duffy, Bernard A., Joliet, Ill.; died October 21; aged 74.

Dunn, Wallace Hays, Winterhaven, Fla.;

University of Pittsburgh, School of Dentistry; died November 6.

Eicholtz, Robert W., Syracuse, N. Y.; Baltimore College of Dental Surgery; died October 14.

Elmore, A. B., Magnolia, Ark.; died September 25; aged 80.

Epes, Travis F., Roanoke, Va.; died October 8; aged 58.

Filkins, Warren V. H., Arlington, N. J.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania; died November 30; aged 68.

Fisher, H. R., Palmyra, N. Y.; died in 1945.

Frank, John P., Elizabeth, N. J.; New York University, College of Dentistry; died November 22; aged 53.

Frank, P. A., Paterson, N. J.; died November 22; aged 63.

Freeman, Carey V., Jacksonville, Fla.; died in 1945.

Gaither, W. D., Memphis, Tenn.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania; died October 15.

Gibbin, Floyd E., Buffalo, N. Y.; University of Buffalo, School of Dentistry; died December 12; aged 54.

Giffin, B. B., Superior, Wis.; Northwestern University Dental School; died October 28; aged 75.

Gifford, Archie C., Oshkosh, Wis.; Marquette University Dental School; died in 1945; aged 61.

Golson, W. H., Fort Deposit, Ala.; died October 10.

Grant, D. S., Sidney, N. Y.; died November 27; aged 44.

Greulich, C. Bert, Cleveland, Ohio; Western Reserve University, School of Dentistry, 1925; died December 7. Dr. Greulich was a lieutenant commander in the U. S. Navy Dental Corps.

Hagen, Paul, Crookston, Minn.; University of Minnesota, School of Dentistry; died December 9; aged 75.

Harms, Richard G., Oakland, Calif.; University of California, College of Dentistry; died in October; aged 77.

Hasbrouck, James F., Pasadena, Calif.; Columbia University, School of Dental and Oral Surgery, 1894; died in 1945; aged 75.

Hazelton, Frank K., Trenton, N. J.; died December 23; aged 75. Dr. Hazelton was secretary of the New Jersey State Dental Society.

Hearn, F. F., Eatonton, Ga.; died October 22.

Helms, William O., Charleston, S. C.; died December 9.

Hendricks, George W., Kingston, N. Y.; died November 12.

Henry, W. G., Punxsutawney, Pa.; Philadelphia Dental College, 1892; died November 23; aged 81.

Hersch, David, Seattle, Wash.; died October 3; aged 57.

Hillier, Charles W., Chicago, Ill.; died November 26; aged 74.

Hinkley, Donald M., Battle Creek, Mich.; University of Michigan, School of Dentistry; died December 10; aged 37. Dr. Hinkley was a captain the U. S. Army Dental Corps.

Hobson, Fred Nae, Griswold, Iowa; died November 16; aged 58.

Hoffman, George I., Spokane, Wash.; died in 1945.

Holland, Le Roy, Dyersburg, Tenn.; University of Tennessee, College of Dentistry; died October 31.

Hopper, A. B., Hastings, Nebr.; died in November.

Hoskins, J. A., Appalachia, Va.; died in March.

Howorth, George T., Lakeview, Mich.; died November 18; aged 82.

Hulse, P. Conner, Palmyra, N. J.; died September 27.

Hussong, George L., Buffalo, N. Y.; University of Buffalo, School of Dentistry; died October 25; aged 86.

Jaeger, Samuel, New York, N. Y.; died June 20.

Jantz, Emil, Walla Walla, Wash.; North Pacific College of Oregon, School of Dentistry, 1913; died October 1.

Jenkins, J. B., Pasadena, Calif.; died November 21.

Jeffries, Richard, Port Jervis, N. Y.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania; died December 1; aged 73.

Johnson, Clayton S., Uniontown, Pa.; University of California, College of Dentistry; died November 12.

Johnson, Ralph, Burlington, Wash.; died October 24; aged 41.

Jones, Aimee L., Louisville, Ky.; University of Louisville, School of Dentistry; died October 25.

Jurissen, Everett P., Yuma, Colo.; died in July.

Kelly, Walter, Memphis, Tenn.; University of Michigan, School of Dentistry; Northwestern University Dental School; died November 23.

Kenny, Charles, B., New York, N. Y.; New York University, College of Dentistry; died December 22.

Keppeler, Edward A., Milwaukee, Wis.; died November 21; aged 55.

Knight, Willard H., Salem, Mass.; died October 22; aged 73.

Knowles, Henry, Falls Church, D. C.; died October 7; aged 79.

Krume, E. L., Grand Rapids, Mich.; Northwestern University Dental School; died September 30; aged 62.

Langan, Thaddeus A., Jersey City, N. J.; Columbia University, School of Dental and Oral Surgery; died in December; aged 55.

Lapinska, Lawrence, Indianapolis, Ind.; Indiana University, School of Dentistry; died November 19; aged 58.

Lasater, John P., Bridgeport, Ala.; died December 8; aged 84.

Learn, Henry Wirt, San Diego, Calif.; State University of Iowa, College of Dentistry; died November 6; aged 84.

Lehrman, Herman, Union City, N. J.; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania; died November 23; aged 66.

Lentz, E. H., Baxter Springs, Kan.; died October 31; aged 58.

Link, Leo E., San Francisco, Calif.; died October 22; aged 45.

Lynch, Thomas A., Los Angeles, Calif.; died in 1945.

McDonald, J. F., Chicago, Ill.; died October 22; aged 65.

McKloskey, Howard Wilson, Rio de Janeiro, Brazil; died November 24.

McKnight, Albert J., Boston, Mass.; died November 7; aged 78.

McMillan, Peter A., Tampico, Ill.; died October 8; aged 72.

Manville, George P., Richmond Hill, Long Island, N. Y.; New York University, College of Dentistry; died October 12; aged 81.

Marlow, George C., San Diego, Calif.; died November 16; aged 81.

Martin, John, Islip, Long Island, N. Y.; died October 31.

Maxwell, George M., Towanda, Pa.; died October 11; aged 70.

Mead, Frank H., Bangor, Maine; Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania; died September 18; aged 75.

Meek, Horace G., Oakland, Calif.; College of Physicians and Surgeons of San Francisco, School of Dentistry; died November 19; aged 52.

Merrill, Elliot, Staten Island, N. Y.; died November 24. Dr. Merrill was a captain in the U. S. Army Dental Corps.

Merwin, Ben S., Walworth, Wis.; Northwestern University Dental School; died November 10; aged 55.

Miller, George Alfred, Pasadena, Calif.; died September 7.

Mindlin, M. H., Lakewood, N. J.; New York University, College of Dentistry; died November 14.

Moderno, Louis, Santa Barbara, Calif.; Tufts College Dental School; died October 10; aged 67.

Moran, Roy, Detroit, Mich.; University of Michigan, School of Dentistry; died October 23; aged 53.

Moreau, Henry L., Prawling, Conn.; died October 30; aged 68.

Morrison, Roderick C., Houghton, Mich.; University of Michigan, School of Dentistry; died October 10.

Neeley, Nathaniel A., St. Petersburg, Ga.; Vanderbilt University, School of Dentistry; died December 13; aged 81.

Newcomer, A. I., Checotah, Okla.; died in October; aged 76.

Nifong, S. C., St. Louis, Mo.; died November 20; aged 69.

Noetzel, Adelbert J., Milwaukee, Wis.; Marquette University Dental School, 1919; died in December.

O'Brien, Walter J., New York, N. Y.; died September 21.

Olsen, Arthur E., Gibbon, Nebr.; Lincoln Dental College, 1917; died October 7; aged 55.

MEETINGS AND ANNOUNCEMENTS

American Academy for Plastics Research, Chicago, Ill., February 10. Sylvester W. Cotter, 11059 South Hale St., Chicago 43, Secretary.

American Academy of Periodontology Council, Chicago, Ill., February 9. Raymond E. Johnson, 824 Lowry Medical Arts Bldg., St. Paul 2, Minn., Secretary.

American Society for the Advancement of General Anesthesia in Dentistry, New York City, March 25, Hotel New Yorker. M. Hillel Feldman, 730 Fifth Ave., New York City 19, Secretary.

American Society of Oral Surgeons, Chicago, Ill., February 7-9. Harry Bear, 410 Professional Bldg., Richmond 19, Va.

Chicago Dental Society, February 11-14. Robert J. Wells, 30 North Michigan Ave., Chicago 2, Secretary.

Dental Hygienists Association of City of New York (first Friday of month from October to May). Grace E. Clinchy, 67-134 Clyde St., Forest Hills 2 A, New York, Publicity Chairman.

Dental Laboratories Institute of America, Chicago, Ill., February 9, Hotel Continental. E. Reed, 8 South Dearborn St., Secretary.

Dental Society of the State of New York, New York City, May 13-16. Charles A. Wilkie, 1 Hanson Place, Brooklyn, Secretary.

Florida State Board of Dental Examiners, Jacksonville, April 8-11. A. W. Kellner, P.O. Box 155, Hollywood, Secretary.

Greater Philadelphia Annual Meeting, February 5-7. Abram Cohen, 269 South Nineteenth St., Philadelphia 3, Chairman, Publicity Committee.

Indiana State Board of Dental Examiners, Indianapolis, May 13-16 (dentistry and dental hygiene); May 14-15 (hygienists). Carl A. Frech, Gary National Bank Bldg., Gary, Secretary.

Iowa State Board of Dental Examiners, Iowa City, March 18-22. Harry G. Bolks, Badgerow Bldg., Sioux City, Secretary.

Iowa State Dental Society, Des Moines, May 6-8, Hotel Fort Des Moines. A. N. Humiston, 417 Higley Bldg., Cedar Rapids.

Kentucky State Board of Dental Examiners, Louisville, March 26-29. W. F. Walz, 1114 First National Bank Bldg., Lexington 3.

Louisiana State Dental Society, Baton Rouge, April 4-6. Julian S. Bernhard, 407 Medical Arts Bldg., Shreveport 45, Secretary.

Maryland State Board of Dental Examiners, Baltimore, March 11-13. Kyrle W. Preis, 700 Cathedral St., Baltimore 1, Secretary.

Massachusetts Dental Society, Boston, May 6-9. Stephen P. Mallett, President.

Michigan State Dental Society, Detroit, April 15-17. H. C. Gerber, Jr., 1514 Olds Tower Bldg., Executive Secretary.

Minnesota State Dental Association, St. Paul, February 25-27. L. M. Cruttenden, 498 Lowry Medical Arts Bldg., St. Paul 2, Secretary.

Mississippi Dental Association, Jackson, May 26-29. O. L. Colee, Magnolia, Secretary.

Nebraska Board of Dental Examiners, Omaha, March 20-23, Creighton College. C. A. Bumstead, Capitol Bldg., Lincoln, Secretary.

New Jersey State Board of Registration and Examination in Dentistry, Trenton, March 25-28. W. A. Wilson, 150 East State St., Trenton.

New Jersey State Dental Society, Atlantic City, May 21-24. J. Ward Weaver, Convention Director, 47 South Main St., Pleasantville.

New York Society of Orthodontists, New York City, March 4-5. Norman L. Hillyer, Professional Bldg., Hempstead, Secretary.

North Carolina State Board of Dental Examiners, Raleigh, April 1. Wilbert Jackson, Rich Bldg., Clinton, Secretary.

Ohio State Dental Board, Cleveland, February 28-March 2 (practical); Columbus, March 11-13 (practical); March 14-16 (theory). Earl D. Lowry, 79 East State St., Columbus 5, Secretary.

Pennsylvania State Dental Council and Examining Board, Philadelphia, March 4-9. Reuben E. V. Miller, 61 North Third Ave., Easton, Secretary.

Pennsylvania State Dental Society, Pittsburgh, May 7-9. C. J. Hollister, 217 State St., Harrisburg, Executive Secretary.

South Carolina State Board of Dental Examiners, Columbia, March 28-30. T. C. Sparks, 1508 Washington St., Columbia 23, Secretary.

South Dakota State Board of Dental Examiners, Sioux Falls, February 28-March 4. T. E. Barrington, P.O. Box 5, Rapid City, Secretary.

Texas State Board of Dental Examiners, Houston, February 18. Carl C. Hardin, Jr., 311 Capitol National Bank Bldg., Austin, Executive Secretary.

Thomas P. Hinman Mid-Winter Clinic, Atlanta, Ga., March 24. J. A. Broach, 1105 Doctors' Bldg., Chairman.

Vermont State Dental Society, Burlington, May 22-23. F. A. Reid, Windsor, President.

Virginia State Board of Dental Examiners, Richmond, March 26, Medical College of Virginia. John M. Hughes, 715 Medical Arts Bldg., Richmond 19.

Washington University Dental Alumni Meeting, February 21-22, Washington University Dental School. V. P. Thompson, 111 West Lockwood Ave., Webster Groves, Mo., Secretary.

West Virginia Kanawha Valley Dental Society Annual Mid-Winter Clinic, Charleston, February 9. R. N. Frame, Charleston, Secretary.

West Virginia State Board of Dental Examiners, West Liberty, June 24-27. R. H. Davis, Clarksburg, Secretary.

Wisconsin State Board of Dental Examiners, Milwaukee, March 11-15. S. F. Donovan, Tomah, Secretary.

Wisconsin State Dental Society, Milwaukee, March 25-27. Kenneth F. Crane, Bankers' Bldg., Milwaukee 2, Secretary.

Internships, Externships, Residencies and Fellowships

Eastman Dental Dispensary, Rochester, N. Y. Director, Harvey J. Burkhart.

Joseph Samuels Dental Clinic of the Rhode Island Hospital, Providence. Ernest A. Charbonnel, Director.

Research and Educational Hospital, University of Illinois Medical Center. Address Dean, University of Illinois, College of Dentistry, 808 South Wood St., Chicago 12.

Richmond Freeman Memorial Clinic for Infants and Children, Dallas, Texas. Dora B. Foster, 3617 Maple Ave., Dallas, Executive Director.

Walter G. Zoller Memorial Dental Clinic, University of Chicago. Address Director, Walter G. Zoller Memorial Dental Clinic, University of Chicago, Chicago 37.

Fellowships for Research and Graduate Work in the Dental Sciences, University of Rochester, George H. Whipple, Rochester, Dean.

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